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INSTITUTO DE MEDICINA TROPICAL

PROCEEDINGS OF THE SIXTH INTERNATIONAL CONGRESSES ON TROPICAL MEDICINE AND MALARIA

Lisbon, September 5 – 13, 1958

Volume VI

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DIVISION A

TROPICAL MEDICINE

(Continued)

SECTION 5

TROPICAL PHYSIOLOGY

SUBSECTION 1

PHYSICAL ENVIRONMENT

1ST SESSION: **Acclimatization to heat**

Tuesday, September 9th — 9:00 to 10:30 a. m. Room A

The meeting was held under the chairmanship of Dr. G. P. Crowden.

The Rapporteur was Dr. C. S. Leithead and the Secretary was Dr. Carlos F. de Oliveira.

The following papers were presented and discussed.

ACCLIMATIZATION OF MEN TO HOT ENVIRONMENTS

SID ROBINSON

Indiana University, Bloomington, Indiana

When unacclimatized men attempt to perform moderate work in a hot environment, they become overheated within 1 or 2 hours, showing markedly elevated rectal and skin temperatures and reduced gradients between internal and surface temperature; metabolism is increased in proportion to body temperature; heart rates are nearly maximal and the men show signs of circulatory instability, the latter being most pronounced in work or when they attempt to stand; exhaustion is imminent. With repeated daily exposures to the same combination of stresses physically fit men acclimatize rapidly — 4 to 8 days. The acclimatization is characterized by (*a*) a gradual improvement in the efficiency of evaporative cooling and in the sensitivity and capacity of the sweating mechanism, (*b*) rapid compensatory adjustments in the renal output of water and electrolytes, (*c*) an increase of circulating blood volume and of interstitial fluid volume during exposure, accompanied by marked improvement in circulatory stability with reduced heart rate, and (*d*) a gradual improvement in temperature regulation so that by the 8th day of exposure, if the heat stress is not too intense, the men may perform the work in the heat with increased gradients between rectal and skin temperature, and with no greater elevations of core temperature and metabolism than when they perform the same task in a cool environment. The improved evaporative cooling of the skin of acclimatized men in the heat is responsible for the increased temperature gradient from core to surface, and thus a smaller cutaneous blood flow suffices to transfer the metabolic heat to the skin. The course and rate of this remarkable transformation of men which takes place during acclimatization to heat may be affected (*a*) by dehydration and/or by salt depletion if they are allowed to occur (both increase circulatory strain and interfere with temperature regulation of men in the heat), (*b*) by the physical fitness of the subjects (fit men adapt more quickly to life in the heat), and (*c*) by their physical activity during the period of acclimatization (sedentary men do not acclimatize fully for work in the heat).

The maintenance of heat balance in the body in hot environments is dependent to a great extent on the circulatory system. The conductance of heat from the tissues in which it is being produced to the skin and respiratory system where it is dissipated is largely dependent on the circulation; simple conductance by body tissues alone would severely limit the rate of heat transfer from the interior to the surface of the body. The blood and tissue fluids, composed largely of water, have a high specific heat and high conductivity of heat and these properties are important in that large amounts of heat can be stored and transported with small changes in temperature.

The capacity of the circulation to make the varied adjustments required in heat regulation in hot environments is surprisingly great. The maximal adjustments possible in a man well acclimatized to work in the heat are illustrated by the following example. In our laboratory acclimatized men have been found to be capable of maintaining thermal equilibrium (rectal temperature 38-38.5°C; heart rate 110-135) while working for 6 hours with metabolic rates of 190 Cal./m^2/hr. in an air temperature of 50°C with relative humidity 18 %, or in an air temperature of 33°C with 95 % humidity (1). In these 6-hour equilibrium experiments the magnitude of cutaneous blood flow is shown by a tissue heat conductance of 78 Cal./hr./m^2/°C difference between rectal and mean skin temperature, or twice the maximal heat conductance of a resting acclimatized man in severe heat, and 8 times the minimal conductance of a man resting in a cool environment (14). When unacclimatized men attempt to work in such heat stresses, they become overheated (rectal temperature 39-40°C; heart sate 160-190) and are exhausted within 2 hous (2-8). The circulatory system of a well acclimatized man working in thermal equilibrium in these situations is meeting a threefold demand in addition to the requirement for the usual metabolic processes: (*a*) a large blood flow through the working muscles provides for respiratory exchange and conducts away the excess heat produced there, (*b*) blood flow through the skin cools the blood and supplies the sweat glands with water at rates averaging as high as 1.5 l/hr. for 6 hours, (*c*) flow through the alimentary tract transfers water from there to the skin and sweat glands. A man is able to achieve this adjustment by increasing the minute volume of his heart to about 2.5 times its resting level and by very fine vasomotor adjustments operating upon a blood volume which may be considerably increased (15 to 20 %) over the volume observed in the man when not adapted to the heat (9-13).

Increased blood flow through the skin is brought about by cutaneous vasodilatation and under ordinary conditions results in increased tissue heat conductance and skin temperature, which in turn increases heat loss from the skin by evaporation, radiation and convection. Selective reactions in different skin areas are a prominent characteristic of cutaneous circulation as shown by Hertzman (15, 16), who found the richness of blood supply to various skin areas to be arranged in descending order as follows: finger pad, ear lobe, toe pad, palm of hand, forehead and face, forearm, knee and tibia. A number of investigators have found that blood flow through the hands and feet is primarily related to body heat regulation. Burton (17) observed simultaneous increases in flow to fingers, toes and nostrils in response to heat stimulus. Scott *et al.* (18) found an increase of blood flow through the fingers from control values of 1 cm^3/min./100 g of tissue in a cool environment to 80 cm^3 or 90 cm^3/min. when the men were exposed to heat. Responses of this type, involving simultaneous dilatation of large vascular areas, are accompanied by changes in general blood pressure and also by changes in heart output and blood volume according to the experiments of Bazett (7).

Bazett's group (9, 18) also observed that 4 days of acclimatization to a warm environment caused both the maximal and average blood flow through a man's fingers to increase by 10 to 25 % over values observed on the first exposure to heat. When exposed to cold after acclimatization to heat, the men were unable, by vasoconstriction, to reduce blood flow to the fingers to the usual low level of about 1 cm^3/100 g of tissue/min. It required 2 or 3 days of exposure to cold to prepare the heat-acclimatized men for maximal peripheral vasoconstriction.

Grant and Pearson (19) made the significant observation that the venous blood flow through the skin of the forearm is derived not only from the digits and hands, but to an important degree from the muscles of the forearms. Since cutaneous veins are thus supplied with blood from the muscles and deeper tissues, they must play an important and direct part in heat regulation. Bazett *et al.* (9) noted increased distention of the superficial veins of the forearm in relation to 4 or 5 days of acclimatization to heat. Bass *et al.* (13, 20) suggest that increased venomotor tone may be an important part of the acclimatization of men to heat. In relation to this, there is evidence of increased peripheral resistance and decreased forearm volume by men in the heat after full acclimatization and these findings are in turn consistent with the fall in skin temperature which has been repeatedly observed during acclimatization (3, 4). Landis has found that changes in venomotor tone play an important part in compensating for blood loss or pooling.

Circulatory stability of acclimatized men working in the heat is facilitated by profound visceral vasoconstriction in compensation for the large cutaneous vasodilatation required in heat dissipation. As evidence of this we have found that renal plasma flow of men working in the heat is reduced by 50 to 70 % from control values observed on the same subjects at rest in a cool environment (21, 22).

The question of whether internal body temperature of resting men is permanently elevated in hot climates was debated by numerous observers for many years (23). More recently observers have found that it probably is not, either in long term residents of hot climates, or in subjects during experimental studies of acclimatization. For example, Burton *et. al.* (24) found that when men unacclimatized to the heat were suddenly exposed to a warm environment (32.4°C dry bulb, 24° wet bulb), their rectal temperature during rest on the first day rose about 0.5°C above the usual level. This shift was gradually decreased in the next few days as adaptation to the warm environment progressed.

The process of acclimatization to work in hot environments involves rapid improvements in the regulation of body temperature (3-8, 25-28). For example, data of Robinson *et. al.* (3) show that when men who were well trained for performing a fixed task of moderate work in a cool environment performed the same work in an air temperature of 40°C and with 23 % relative humidity, their rectal and skin temperatures were raised to averages of 39.7°C and 37°C respectively and their heart rates to 180 within 60 to 90 minutes. Daily repetition of the same task in the same hot environment made it possible within 4 to 8 days for them to perform the work without distress, with mean skin temperatures reduced to 35.8°C and rectal temperature elevated only to the control levels they had attained in performing the same work in a cool environment. Men's mean skin temperature decreases more than rectal temperature during acclimatization to work in the heat, indicating a more effective evaporative cooling of the skin and a reduction of blood flow to the skin (3, 4). Marked reductions of heart rate indicate decreased strain on the circulation (3-7).

Knipping (29) found that the basal metabolism of healthy individuals increased during the first week of exposure to a hot climate, that it then fell gradually and after several months' residence in the hot climate was below the ordinary basal standards. Burton (24) also found that men unacclimatized to the heat underwent a moderate rise in metabolism during the first few days in a hot environment. He attributed the rise to elevated body temperatures and found that, after 5 days acclimatization, rectal temperatures and metabolism both returned to control levels. There is some evidence that the basal metabolism of men may be reduced by residence in hot climates and that this is true for natives as well as for Europeans who have moved to the tropics. Numerous investigators (29-34) have found evidences of reduced metabolism in residents of hot climates, reductions ranging from 6 to 24 % having been reported. On the other hand, Eijkman (35) found Europeans in the Dutch East Indies who underwent no lowering of metabolism.

In unacclimatized men performing work at a constant rate in a hot environment (dry bulb 40°C, relative humidity 23 %) we have found that metabolism is increased by about 11 % for each degree centigrade elevation of rectal temperature above control values in performing the same work in a cool environment. These data on men walking on the treadmill at 5.6 km/hr. up a 5.7 % grade confirm the observation by DuBois (36) on resting fever patients that the elevation of metabolism accompanying increased body temperature is according to van't Hoff's law. After 1 to 3 weeks of acclimatization, metabolism of the men performing the same work in the heat returned to the control level, because they had become able to do the work in the heat with no greater increases of rectal temperature than those they experienced in a cool environment (3).

Dehydration, whether it be primary or secondary to salt depletion, results in significant reduction in circulating plasma volume (6, 14). Reduced plasma volume in dehydration leads to circulatory instability and failure of temperature regulation by men in the heat (5, 6, 14, 37), whereas an expanded blood volume is required to maintain circulation of men in the heat (9-13, 20). Thus maintenance of water balance, which can be achieved only if *NaCl* balance is also maintained, is a necessity for rapid acclimatization to heat. Attempts of salt-depleted men to keep up body hydration by forced drinking of water may result in extracellular dilution with diuresis and secondary dehydration (38, 39). Thirst, dependent on increased extracellular *NaCl* concentration (40), is therefore not an adequate indication of dehydration in a salt depleted man (6, 37-40). From these statements it is obvious that men can pass through the stages of acclimatization to heat with minimal disturbances of temperature regulation and circulatory stability if they regulate their intakes of water and salt to balance losses through kidneys and sweat glands, plus enough extra to provide for a 15-25 % isotonic expansion of the extracellular fluid volume.

The kidneys have as their primary function the maintenance of chemical homeostasis. In this role they are largely under endocrine control (adrenal cortex and posterior pituitary) and are capable of making rapid alterations in their output of water (10-400 cm^3/hr.)

and of salt (0-20 mEq/hr.) in response to deficiencies or excesses of these substances in the body (38-42). In deficiencies of water and electrolytes the kidneys are able to conserve these materials, but the deficiencies can be corrected only by adjusting the intake to exceed the rate of loss. Bass (13, 20) credits the kidneys of men with the ability to conserve salt and water in hot environments in response to the obvious need for expanding the extracellular fluid volume. He suggests volume receptors, related to hemodynamic changes, as a possible mechanism of this response in the heat.

The sweat glands, in contrast to the kidneys, are primarily adapted for evaporative cooling of the skin in temperature regulation and are under thermostatic nervous control (14, 43-47). In acclimatized men, sweating responses to thermal stimuli and to work are obligatory, rapid, extremely sensitive and precisely adjusted to the body's need for evaporative cooling (14, 43-49). In men under thermal and work stresses, sweating will continue to extract water and *NaCl* from the body even in the face of significant deficiencies of these substances (38-42, 51-56). As men acclimatize to work in extreme heat their maximal capacities for sweating may increase by 30 to 50 % in three weeks (3-5, 8, 25, 27) and the *NaCl* concentration in sweat may be greatly reduced (37-42, 50-56). On the other hand, where the heat stress is moderate, the evaporative requirements of men for thermal equilibrium become lower as their efficiencies for the work improve with training and with reduced body temperatures resulting from acclimatization (3, 14). The reduction of sweat concentration contributes importantly to the maintenance of water and electrolyte balance of men in the heat.

The following example will illustrate the differences between the functions of sweat glands and kidneys of men in their adjustments to heat. If the *NaCl* loss in sweat exceeds the intake of men during exposure to heat, the kidneys quickly reduce their salt output and in 5 hours the urine may be free of salt, whereas the sweat glands reduce their salt concentration slowly, requiring several days to complete their adaptation to a salt deficiency (39-42, 51-56). The slow salt-conserving responses of the sweat glands occur in response to salt deficiency involving a reduction in the salt concentration of extracellular fluid. The kidneys of men exposed to heat conserve both salt and water in response to deficiencies of these substances (39-42) and also to a need for isotonically expanding the extracellular fluid volume in order to re-establish cardiovascular stability (13). During initial exposures of unacclimatized men to heat involuntary dehydration may result from high sweat rates and a suppression of thirst, associated with work and/or with salt deficiency (6, 13, 14, 37, 42). The kidneys respond rapidly to such dehydration by reducing urine flow to 10 or 12 cm^3/hr. The rates of sweating of men working in the heat are reduced about 20 % within an hour by water deficits of only 1 % of body weight (38, 57), but in the author's experience deficits up to 4 % do not further reduce sweating (38). If salt balance is maintained, or when salt deficiencies are made up, thirst will demand rapid replacement of water deficits, especially during periods of rest.

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HUMAN WATER ECONOMY IN THE HEAT

W. V. MACFARLANE

Man sweats to survive in the tropics. This involves short and long term adjustment of fluid spaces, endocrine and renal changes, and modified cell functions. Some of the time sequences of adjustment have been examined in both the field and the laboratory.

DAY-NIGHT CHANGES

Water lost by sweat during the day is not replaced fully until late in the evening. The lag of water intake behind water loss in adult workers in the tropics reaches 2-3 l. Urinary excretion of water and *Na* is less in the 6 A.M.-6 P.M. period than in the 12 night hours, in the tropics. The *K/Na* ratio is high by day and low at night.

ACUTE HEATING

After 4 hours at 40°C (during which 2-3 l sweat may be produced and both *Na* and water loss by kidneys are reduced) there is a diminished extracellular volume, often associated with increase of plasma volume. Aldosterone increases rapidly in the urine on sweating and results in *Na* retention, *K* excretion. The 17-ketogenic steroid fraction of urine decreases during heating while urinary aldosterone increases. On cooling there is a *Na* diuresis, delayed 24 to 48 hours.

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LONGER PERIODS OF HEATING

Water intake is proportional to ambient temperature when the work done is constant.

Fasting body weight is reciprocal to shade temperature, and weight changes proportionally to urinary water and sodium output. There is a lag of 2 days after cooling before sodium and water accumulations are excreted.

SEASON

The average urinary output of 17-ketosteroid and 17-ketogenic steroid is 30 % higher in winter than in summer, while the urinary *K/Na* ratio (which is an index of aldosterone-like activity) is high in summer, low in winter, in adults on lat. 27° S. Antidiuretic substance (ADS) in plasma appears only when plasma is concentrated, and this occurs in summer but not in winter. The seasonal difference appears to arise from the availability of water to plasma during winter, even though sweat loss be identical at all seasons.

The function of ADS in relation to heat appears to be the prevention of water diuresis in summer, during heavy sweating. Evidence that the plasma material reducing urine flow in alcohol-treated rats is vasopressin (ADH) from the pituitary is derived from the following observations:

a) The time course of antidiuresis in assay rats is the same for plasma ADS pituitary ADH (vasopressin);

b) Concentration-action curves for plasma ADS and ADH are parallel;

c) Equal amount of ADS and ADH are inactivated by the same amount of thioglycollic acid.

There is evidence, however, that ADH or ADS is not the main regulator of water loss from the kidneys during sweating:

a) Urine flows of less than 0.5 ml/min. are induced by heating in summer several hours before a change of ADS concentration is detected in plasma. In winter no ADS is detected in spite of low urine flows in heated subjects;

b) Intravenous administration of 1000 μU of ADH (raising the plasma ADS to 300 μU/ml or more) fails to reduce urine flow below 0.5 ml/min., whereas in men sweating 800 ml/hr. urine flow falls to 0.1-0.2 ml/min.

Whether neurovascular adjustment of kidney function or a direct plasma action on renal cells or some unknown mechanism is responsible for low urine flow in the heat is not yet determined.

NATURAL ACCLIMATISATION TO HEAT

CHARLES S. LEITHEAD
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Throughout the summer months of 1956, the pulse rate, blood pressure and subjective responses to exercise and to sudden postural change were measured in 24 men from the United Kingdom during their first week in the desert climate of Kuwait, Arabia. Packed blood cell volumes (haematocrits) and plasma protein levels were also estimated. All 24 men, in groups of 3 or 4, had travelled by air from England to Kuwait to take up employment with the Kuwait Oil Company. They varied considerably in age (from 19 to 40 years) and in physical fitness; some were administrators or office-workers, others were engaged as drillers or similar production operators in the desert. As they were not required to begin work until one week after arrival, their activities during this period were largely self chosen, some spending their leisure hours in idleness, while others indulged in tennis, swimming, or other forms of exercise.

The purpose of this investigation was to observe a few simple physiological and psychological reactions in men exposed to unaccustomed environmental heat; it was not designed to assess their progress of acclimatisation by the usual indices. Sweat rates, rectal temperatures and blood volumes were not measured.

MATERIALS AND METHODS

Reactions to postural change were investigated by use of a tilt-board or table which could be swung rapidly and easily into a position 75° to the horizontal; a foot-rest was provided to obviate

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the need for retaining or safety-belts. The pulse rate and blood pressure of each subject were measured after 3 minutes rest in the horizontal position, immediately after tilting, and finally after 3 minutes in the 75° position.

The subject was then required for a period of 16 seconds to step on and off a platform 18" high at a standard rate and according to a predetermined pattern, pulse rates being measured before the test and at 15 second intervals for 2 minutes afterwards. Information obtained from the tilt-table and from the stepping test was scored according to Schneider's (1924) method in order to provide an index of cardio-vascular fitness. Fourteen British volunteers who had been resident in Kuwait for some years acted as control subjects.

Packed blood cell volumes were estimated by the Wintrobe method, and plasma proteins by the Van Slyke copper sulphate specific gravity technique. The estimations were made on the first day of exposure and again on the fourth day; in 9 cases, the haematocrit and plasma protein levels were re-examined 2 weeks later.

Room temperature in the laboratory where the tests were performed was kept standard at (dry-bulb) 25°C. The average outdoor daily temperatures in Kuwait during the experimental period were dry-bulb 44°C and wet-bulb 25°C.

RESULTS

It was found that, of the 24 subjects tested, 14 had never before been exposed to hot climates. This group appeared to adapt less readily to their new environment, and 2 of them developed prickly heat and dysentery and were consequently unable to begin work at the appointed time. Moreover, the only subjects to experience giddiness or syncope (Table 1) as a result of their first tilt-table test were members of this group. The remaining 10 subjects, on the other hand, adapted well and had no subjective reactions to tilting, although in all but 2 of the cases the period elapsing between previous and present exposure to heat was not less than 1 year, and in 2 others it was as much as 6 years. The duration of previous exposure to heat was in no instance less than 2 years.

TABLE 1 — *Subjective responses to first tilting*

Group	Number of subjects	Giddiness only	Immediate syncope	Delayed syncope
C.	14	—	—	—
A (I)	10	—	—	—
A (II)	14	4	1	3

C : Resident control subjects.
A (I) : Acclimatisers with previous tropical experience.
A (II) : Acclimatisers without previous tropical experience.

A summary of pulse rate and blood pressure responses to first tilting (Table 2) shows that, in the group of acclimatisers with no previous tropical exposure, those who experienced giddiness or syncope on first tilting otherwise behaved in no way differently from their associates who tolerated the manoeuvre well. The circulatory reactions of acclimatisers with previous experience of hot climates were similar to the reactions of the resident control subjects.

TABLE 2 — *Mean circulatory responses to first tilting*

Group	Number of subjects	Pulse rate			Systolic B.P.			Diastolic B.P.		
		Resting	Initial upright	Final upright	Resting	Initial upright	Final upright	Resting	Initial upright	Final upright
C	14	70	80	81	112	114	114	75	86	83
A (I)	10	70	80	84	112	114	115	72	86	86
A (II)	8	74	93	83	108	104	107	70	86	85
A (III)	6	68	88	90	109	103	108	70	82	84

A (II) : Acclimatisers with no previous tropical experience who had giddiness or syncope on tilting.
A (III) : Acclimatisers with no previous tropical experience and with no subjective sensations on tilting.

On the second and subsequent days of exposure to heat, there were no significant differences in responses to tilting between the subjects with previous tropical experience and those without, and after the first test there was no giddiness or syncope in either group. Haematocrit and plasma protein levels were reduced by the 4th day of exposure in all but one of 18 subjects so investigated. Nine of the 24 attended the laboratory on 5 occasions — on the first, second, fourth and fifth days of exposure and again 2 weeks later. The information obtained is summarised in Table 3 and in Table 4, in which are included Schneider indices, haematocrits, and plasma proteins of the same subjects. The Schneider scoring system allowed for a possible maximum of + 18, but 14 resident control subjects on 80 occasions achieved an average of + 12. Haematocrit and plasma protein figures obtained on the fourth day are significantly lower than on the first and final days.

TABLE 3 — *Mean circulatory responses to tilting of 9 subjects exposed to environmental heat*

Days of exposure to heat	Pulse rate			Systolic B.P.			Diastolic B.P.		
	Resting	Initial upright	Final upright	Resting	Initial upright	Final upright	Resting	Initial upright	Final upright
First	73	88	68	109	106	107	76	87	89
Second	73	87	89	107	107	109	68	84	84
Fourth	72	87	86	109	110	112	67	83	81
Fifth	73	85	87	105	106	106	68	82	83
2 weeks later . .	66	74	75	116	119	116	68	87	82

TABLE 4 — *Progress of 9 subjects on exposure to heat*

Mean indices	Days of exposure				
	First	Second	Fourth	Fifth	2 weeks later
Rise in pulse rate (per min.) on tilting	15	14	15	12	8
Change in systolic B.P. on tilting	− 3	0	+ 1	+ 1	+ 3
Schneider fitness index	+ 7.6	+ 8	+ 8.6	+ 10	—
Haematocrit (percent)	47.4	—	43	—	46.3
Plasma protein (grams percent)	7.6	—	7.2	—	7 5

DISCUSSION

Tilt-table and step-test results show signs of circulatory instability in unacclimatised men on first exposure to heat, with a certain, though ill-defined, advantage to sujects with a history of previous experience of hot climates. Improvement in circulatory responses to tilting and to mild exercise was observed throughout the first 2-3 weeks after arrival in Kuwait.

Absence of measurable differences between subjects who were giddy or faint on first tilting and those who were not is in accord with the findings of Allen, Taylor and Hall (1945) and of Macpherson (1949). These subjective symptoms, however, were confined to individuals without previous tropical experience, and may have been related to the psychological stresses imposed on this group, to which everything — climate, surroundings, customs — was completely strange.

The assumption that changes in haematocrit or plasma protein levels reflect changes in plasma volume has been condemned because it takes no account of possible changes in circulating protein or red cell population. In field investigations such as the present one, however, there are often no alternative methods so readily handled by a mobile laboratory or so acceptable to volunteer subjects.

Bass *et al.* (1955) observed that in 5 subjects living in a room heated to 49°C, the extracellular fluid and plasma volumes expanded by the fifth day; total circulating protein increased, but red cell volume did not, and the authors concluded that the «blood volume was increased as a result of fluid which was almost identical with plasma». It is believed that haematocrit values obtained in the present study show an increase in plasma volume on the fourth day of exposure to the summer heat in Kuwait; this occurred in all but one of the 18 subjects so investigated, and was not influenced by previous tropical experience. Similar evidence was obtained in June 1958, when a series of haematocrits of 2 subjects also showed a fall on the fourth day in Kuwait (Table 5).

TABLE 5 — *Haematocrits of 2 subjects, 1958*

Subjects	United Kingdom Baseline		Kuwait-days of exposure to heat					
			3	4	5	6	8	12
K. A. F.	50	49	49	47	46	46	48.5	49
C. S. L.	52	52	52	47	47.5	47.5	48.5	50

The fall in plasma protein levels also observed on the fourth day in Kuwait (Table 4) suggests dilution of the plasma with no concomitant increase in circulating protein, a finding which is at variance with that of Bass and his colleagues.

SUMMARY

In the summer of 1956, the circulatory and subjective responses to heat were studied at Kuwait in 24 men during the first week after their arrival by air from England; 9 of them where re-investigated 2 weeks later. Sudden postural change caused giddiness and syncope only in those who had never before been in hot climates. Pulse rate and blood pressure reactions to tilting and to exercise improved steadily throughout the first week, at the end of which all but 2 of the subjects tested commenced work. A fall in haematocrit and plasma protein levels was demonstrated on the fourth day of exposure, which suggests an increase in plasma volume as a result of simple dilution.

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ACKNOWLEDGEMENTS

I wish to express gratitude to Sir Philip Southwell, Managing Director of the Kuwait Oil Company, for facilities to carry out this research.

THE INFLUENCE OF THE BRAIN ON ACCLIMATIZATION

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If one surveys the work done in recent decades on the regulation of body temperature, one cannot help concluding that we have been thinking too much about effects and too little about causes, too much about peripheral phenomena and too little about the brain. Perhaps this was inevitable, because peripheral phenomena, such as sweating, blood flow, or renal excretion, can be studied more easily than the brain, but the time has come perhaps to restore the balance and to consider the part played by cerebral control.

In a session devoted to acclimatization it may seem wrong to mention immediate responses to changes of temperature; but we can only understand acclimatization if we understand immediate responses, especially since both depend to some extent on the storage of information (Glaser & Newling, 1957, *J. Physiol.,* **137**: 1; Glaser & Whittow, 1957, *J. Physiol.,* **136**: 98; Glaser, Hall & Whittow, 1959, *J. Physiol.,* **146**: 152). It may seem even less justifiable to consider acclimatization to cold at a Congress devoted to the Tropics, but cold acclimatization is inseparable from acclimatization to heat, as will be seen below, and it is more important to people visiting the Tropics than is often realized.

It cannot be denied that some phenomena of acclimatization are purely peripheral and possibly independent of any central nervous control. The thickening of the skin which takes place both in very hot and in very cold countries is perhaps one such purely peripheral

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phenomenon. It must be equally conceded, however, that some changes accompanying acclimatization can only be cerebral in origin. There is evidence, for example, that people who are even partly acclimatized to heat cease to feel discomfort in a hot environment, though their skin temperatures and rectal temperatures are only very slightly lower than before partial acclimatization (E. M. Glaser, 1949, *J. Physiol.*, **110**: 330). There is evidence also that people who are permanently in a hot environment cease to mind being hot (Glaser, 1957, *Med. J. Malaya*, **11**: 164), though their deep and superficial body temperatures can sometimes be very high. It seems possible, further, that a reduction of activity and of unnecessary effort takes place after heat acclimatization, and this may account for much of the improved ability to withstand heat among people who have lived in hot places for long periods. Changes of sensations or of activity are, of course, more likely to be brought about by the brain than by any other mechanism, and there is now some objective evidence that peripheral receptors are not inhibited by repeated warming and cooling, though sensations diminish (Glaser, Hall & Whittow, *loc. cit.*). It is well known also that a measurable degree of acclimatization can sometimes be demonstrated after a single day in a hot room or in a hot climate, and it is improbable that such rapid changes should be brought about by anything other than the central nervous system. Observations made on «local acclimatization» of a single hand (Glaser & Whittow, *loc. cit.;* Glaser, Hall & Whittow, *loc. cit.;* Glaser, Hall & Whittow, 1957, *J. Physiol.*, **140**: 42 P) are clearly compatible with the conclusion that retained adaptations of the circulation in response to cooling or warming of a hand are due to the brain. That being so, it would be far-fetched, indeed, to suggest that the brain can play no part in ajustments of the circulation which take place when the whole body is acclimatized, but irrefutable evidence for this is not yet available.

As already implied, immediate heat regulation and acclimatization merge imperceptibly. Indeed, at any one moment the temperature of the body depends not only on the heat load at that moment, but to some extent also on the preceding heat load (Glaser & Newling, *loc. cit.*). The responses to any given set of environmental conditions will also be modified by previous experience of the same conditions, and this is acclimatization. The dependence of responses upon present and past stimuli is, of course, not confined to temperature regulation, and it has its analogies in learning, in conditioned reflexes, and in habituation, which are all cerebral phenomena based on the ability of the brain to analyse sensory impulses in the light of stored information, in order to produce an integrated response suited to maintain the life and efficiency of the organism. The basis of such cerebral activity is perhaps the variability of transmission of impulses between different cells of the brain; its main elements are the storage of information over short periods by impulses running around reverberating circuits of neurones, the storage of information over longer periods by the formation of fresh neuronal paths through which a given sensory impulse will be canalised, and the modification of such neuronal activity both by other sensory impulses reaching the brain and by existing (or «spontaneous») cerebral activity. This problem has been recently reviewed elsewhere (Glaser, 1958, *Lond. Hosp. Gazette.*, **61**, Suppl.).

It seems obvious from the above that temperature regulation and acclimatization, like present experiences and memories, are continuous and interdependent. It must appear equally obvious that acclimatization to heat and to cold also form part of a continuous process. When hot summers are followed by cold winters, as happens in many parts of the world, or when people go from hot places to cold ones, they do not merely lose their acclimatization to heat, but they acquire acclimatization to cold. The loss of any one acclimatization is thus tantamount to the acquisition of another. Experimental evidence for this was provided nearly ten years ago (Glaser, 1949, *loc. cit.*).

A distinction must be made between the state of acclimatization and the ability to acclimatize. It seems possible that people who remain for long periods at a fairly constant temperature, as for example at sea level near the Equator, are less able to acclimatize to a new environment than people who are used to changes of climate or weather. In other words, the state of heat acclimatization may be good among Equatorial people, but their ability to acclimatize is poor. There is, moreover, experimental evidence to suggest that changes of temperature improve the ability to acclimatize (Glaser, 1949, *loc. cit.*). All this looks as if the body could learn and forget how to acclimatize. Learning and forgetting are, of course, cerebral phenomena, and this variability of acclimatization is again probably due to the brain.

The fact that frequent changes of temperature enhance the ability to acclimatize is an advantage to people who must often move from one climate to another. But the question arises whether it is possible to be acclimatized to heat and to cold at one and the same time. Insofar as acclimatization is brought about by the brain this would seem probable, because it is possible for a wide variety of skills, or conditioned reflexes, or habituations, to exist simultaneously; and it is well within the power of the brain to retain at one and the same time the ability of responding optimally to two apparently conflicting sets of afferent impulses. There is evidence, indeed, that a single hand can be kept adapted to immersions in very cold and very hot water and that it can retain this local acclimatization to heat and cold for periods of up to two weeks (Glaser, Hall & Whittow, 1958, 1959, *loc. cit.*). Though at this moment there is no very definite evidence to that effect, it seems possible that not only hands but people could be acclimatized to heat and to cold at one and the same time, and that they could maintain such a state of acclimatization so long as they were subjected to both heat and cold at intervals of a few days.

GENERAL DISCUSSION

Dr. D. E. Bass (U.S.A.) — I wish to emphasize that I do not deny the occurrence of an increased blood volume during sedentary living in the heat, but rather to state that increased basal blood volume is not a necessary factor in acclimatization to work in the heat.

Dr. W. V. Macfarlane (U.S.A.) — Work in a cool environment can expand the T1824 space 20 % or more. Heat and work together may expand it a little more.

At the end of a hot day, man may, even though taking fluids, have a smaller T1824 and SNC space than initially. But on cooling the T1824 space expands with fluid replacement completed.

Sheep in the tropics have higher SCN and T1824 spaces in both summer and winter than those in temperate regions. They may be a better measure of fluid space responses than man who imposes artificial environments on himself in the tropics.

Dr. F. H. Bonjer (Holland) — It has been pointed out by Dr. Robinson that energy expenditure is lower after acclimatization. Now it sounds very likely that efficiency increases as acclimatization goes on, but is there any explanation for the initial rise in metabolic rate?

Dr. S. Robinson (U.S.A.) — In general the increased metabolism of unacclimatized men in the heat is probably the result of elevated body temperature. DuBois found this to be true in fever patients and we have found that even highly acclimatized men working in extreme heat have work metabolism increased about 10 % for each °C, elevation of rectal temperature above control values in the same work in a cool environment. The high efficiency of the cotton field workmen reported by us in the *Journal of Tropical Medicine*, 1941, was probably due both to the relatively low body temperatures of the men during the work experiments and to their high degree of training and fitness for the task. Their daily work in using walking cultivators prepared them well for work on the treadmill in these experiments.

Tuesday, September 9th — 11:00 to 12:30 a. m. Room A

The meeting was held under the chairmanship of Dr. W. P. Stam.

The Rapporteur was Dr. C. S. Leithead and the Secretary was Dr. Carlos F. de Oliveira.

The following papers were presented and discussed.

HEAT TOLERANCE

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«Heat tolerance» is a deceptively simple title for a very complex subject. Heat in this sense does not imply merely the temperature of the air, but includes also the radiant heat load and the effects of humidity and air speed as well. Indeed, it has been recognized at least from the time of Haldane (1905) onwards that the humidity may be more important than the air temperature in determining the tolerability of a hot environment. This follows inevitably from the fact that the regulation of body temperature in hot conditions depends upon the evaporation of sweat.

I may add in passing that it is very satisfying to observe that no less than two papers in this session are to be devoted to this subject.

To add to the complexity of the subject, the effects of the physical factors just enumerated are profoundly modified by other factors which are related to the subject within the environment. These latter include not only the rate of energy expenditure and the type of clothing worn, but also more remotely associated factors, such as the water intake and salt balance, and by even more remotely asso-

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ciated factors, such as the age, sex, bodily habitus, health and nutrition of the person concerned.

THE MEASUREMENT OF HEAT STRESS

The multiplicity of the factors involved makes difficult the assessment of the heat stress imposed by any specified environment, and it means too that equal tolerability or intolerability may be achieved by combinations of widely different levels of the several factors concerned. These two facts have resulted in many attempts to combine in a single index the effects of two or more of these factors or, ideally, to find an index which will embrace the effects of them all, including those appertaining to the subjects within the environment as well as those relating to the environment itself.

To some it would seem that, in view of the complexity of the subject, the search for such an index is but a modern version of the search for the philosopher's stone, mere wishful thinking inevitably doomed to disappointment. To others it seems that the search must be continued in the hope of satisfying a pressing need. That there is such a need is, I think, unquestionable, if only to provide a comprehensible definition of heat tolerance.

The simplest index of this nature is provided by the wet-bulb thermometer. It takes some account of two components — air temperature and humidity. The wet katathermometer advances one stage further and embraces the effects of air movement as well. These instruments, however, provide measures of the change in the environment which are only indirectly related to the change in the physiological strain experienced by the subjects within the environment. A more direct approach to the problem of assessing the physiological strain produced is to use carefully-trained subjects as measuring instruments.

One of the earliest and most sucessful examples of this method was the devising of the Effective Temperature Scale by Houghten, Yaglou, Miller and Lincoln at the Research Laboratory of the American Society of Heating and Ventilating Engineers in co-operation with the U.S. Bureau of Mines (Houghten and Yagloglou, 1923; Houghten and Yaglogou, 1924; Yagloglou and Miller, 1925).

It is now 35 years since this scale first came into use, and during this time it has come to be appreciated that it has very definite limitations in its application and is even in error in certain respects. It remains, nevertheless, the most widely used scale of physiological effect and its continued use by engineers and others provides perhaps the best proof of the need for such a scale.

The Effective Temperature Scale is too well known to need any detailed description, but there are some points to which attention may well be drawn. It is a scale of physiological strain. It defines the effective temperature of any given combination of dry-bulb temperature, wet-bulb temperature and air speed as the temperature of saturated still air in which the same degree of comfort or discomfort is experienced as in the environment in question, and the comfort and discomfort of a well-trained observer can legitimately be accepted as a measure of physiological strain.

A more fundamental approach to the problem of the measurement of heat stress than the use of the «human yard-stick» would be to consider the man in relation to his heat exchange with his environment. This can be determined from established physical laws and can be expressed in quantitative terms. If the heat loss required to maintain equilibrium exceeds that possible in the circumstances, then clearly the environment is intolerable. Furthermore, if we are prepared to set limits to the use of the emergency mechanisms of heat storage, it is possible to define the time limit for exposure to a given intolerable climate. A further advantage of this method is that it draws attention to the importance of time as one of the parameters of tolerability. The tolerance of an environment can only be assessed in terms of the duration of the exposure. Within limits, any environment is tolerable for some length of time, though in many cases this may be impractically short.

An index based on these principles has been developed (Belding and Hatch, 1955) and my personal views are that this approach represents our best hope for a successful solution to this intractable problem. It is with great interest, therefore, that I look forward to the paper which is to be presented on this topic.

On the whole, the measurements of the physical components of the environment do not present any great difficulties. Air temperature and humidity are readily determined. The measurement of air speed is somewhat more difficult, especially at the low speeds and high degree of turbulence usually encountered, but the measurement of the radiant heat load does present special problems. Indoors, with relatively uniform surroundings, radiating at a reasonably constant rate at low intensities, simple measurements, such as the determination of the mean radiant temperature by means of the globe-thermometer, will usually suffice. It is not so easy, however, to determine the effect of localized radiation from small sources of high intensity or when it fluctuates rapidly, for example when furnace doors are opened or large crucibles containing molten metal move rapidly from place to place. The most important source of radiant heat in the tropics, strong sunlight, provides problems of measurement of the highest order. Valuable progress has been made by the introduction of special instruments such as the pan radiometer (Richards, Stoll and Hardy, 1951), but on account of the changes in altitude of the sun and variations in the profile of the subject exposed the determination of the solar heat load on man and animals in the tropics remains an outstanding problem.

THE MEANING OF TOLERANCE

It is necessary to be very clear what we mean when we say that this or that environment is tolerable or intolerable, as the case may be. The position is more complex than it appears at first sight. If the man exposed to the conditions in question is rendered unconscious, or otherwise completely incapacitated by some form of acute heat illness, then clearly the limit of physical tolerance has been reached and passed. The endpoint is precise and readily determined, but provides information about only one aspect of heat tolerance.

In practice the value of this information is restricted by the fact that long before this physical endpoint is reached the man's powers of perception, reasoning and judgement will be impaired. This sets a lower limit of tolerance which may be described as the mental limit. Unlike the physical limit, however, this cannot be precisely defined as there tends to be a decline with increasing stress (Mackworth, 1950), and it is difficult to decide at what stage the deterioration in performance is significant. An added complication is that, as the limits of tolerance are approached, the subject, as in anoxia, is often unaware of the deterioration in his capacity.

There is also a third, and by far the most important, upper limit to heat tolerance which for lack of a better term may be described as the emotional limit. Long before there is measurable deterioration in either physical or mental performance an increasing degree of discomfort will be experienced. Such discomfort may temporarily be disregarded but sooner or later will prove intolerable and an attempt will be made to escape to cooler conditions. Discomfort will not willingly be endured indefinitely.

In defining tolerable levels of warmth it is necessary to distinguish between these three levels. It is important to know the limits of physical endurance because in crises, especially in war, it may be necessary to employ men in conditions approaching those in which their continued existence may be imperilled. The identification of conditions which are physically tolerable for fit young men for a brief period is, however, much less important than determining the conditions which are acceptable to a whole population for long periods of time. In setting the latter limits it is clearly the zone of comfort which must be determined.

It is clear too that in determining the levels of tolerance the question of time must be considered. If thermal equilibrium cannot be achieved, then clearly the environment is tolerable only for a brief period. But it is not necessarily true that environments in which equilibrium can be achieved are tolerable indefinitely. Compensating mechanisms become fatigued and with the passage of time equilibrium may be lost. The importance of this point is that repeated exposures to high levels of stress may be possible if intervals of recovery in cooler surroundings are permitted, whilst exposure to a lower level of stress may prove disastrous if it is continuous. Continued exposure over a period of years to a level of stress which never exceeds that of mere discomfort may be the basis of the so-called tropical neurasthenia.

The specification of acceptable limits of warmth is made difficult by the variation in tolerability that exists not only between persons, but also in the same person from time to time. Undoubtedly the most important factor concerned in the variation of the subject is his existing degree of acclimatization. A very considerable degree of adaptation to heat can be produced by the process of acclimatization, and it is necessary to bear in mind the probable degree of acclimatization of the population concerned whenever recommendations are made on the subject of permissible levels of warmth. Conditions which are quite acceptable to the well acclimatized may prove disastrous for the newcomer. This applies not only at the level of physical endurance, but also at the level of the comfort zone.

The specification of permissible levels of warmth — a duty often demanded of the climatic physiologist by industry and the armed forces — is fraught with difficulty. One has to give consideration to so many factors — the probable rate of energy expenditure, the clothing, age, sex and health of the persons concerned, to mention but a few — but even when all these factors are taken into account, there remains the variation in tolerability inherent in each individual. Individuals otherwise indistinguishable vary widely in their capacity to withstand the effects of heat.

One must expect the occasional failure in the apparently inoccuous environment and the unexpectedly dramatic survival of conditions that should inevitably prove fatal.

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PROBLEMS IN DEVELOPING A VALID INDEX OF HEAT STRESS

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You would like to know whether a certain job involves dangerous exposure to heat, or how hot the weather was at home yesterday, or how much hotter Calcutta is than Washington, D.C. Such questions could readily be answered if there were a valid method of equating the factors which determine heat stress in terms of the strain which is imposed on human mechanisms. Lacking such means for assessment, we must persist in itemization of dry and wet bulb temperatures, cover, and wind for haphazard integration by the user on the basis of his experience.

The formulation of a suitable heat scale which takes into account the variables of consequence has proven a challenging task, as more than a score of biologists and thermal engineers will testify. Some of the proposals are listed in Table 1. We cannot attempt here to describe or criticize these individually. We can say that all are more or less useful for certain purposes and that none are universally applicable. The deficiencies relate to the fact that it has not yet been found possible properly to take into account the 10

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[1] The author gratefully acknowledges the collaboration of Prof. Theodore Hatch, Dr. Marvin L. Riedesel, Mr. Bruce Hertig and the several subjects who, as part of the Pittsburgh team, have contributed to the observations reported here. The work has been partly supported by the National Institutes of Health and the Quartermaster Research and Development Command.

variables which affect the heat stress — in terms of at least three major foci of human strain [1].

Some systems proposed for relating warm thermal conditions as they affect man

System	Source	Reference
Wet Bulb Index	Haldane *et al.*	1905, *J Hyg.*, **5**, 494.
Katathermometer	Hill *et al.*	1916, *Phil. Trans. Roy. Soc.*, B, **207**: 183.
Effective Temperature	Houghten, Yaglou ASHVE	1923, *Trans. Am. Soc. Heat. Vent. Engrs.*, **29**: 163.
Operative Temperature [1]	Winslow, Gagge *et al.* Pierce Laboratory	1937, *Am. J. Physiol.*, **120**: 1.
Fort Knox Coefficients [1]	Nelson, Hatch *et al.*	1947, *Am. J. Physiol.*, **151**: 626.
Index of Physiological Effect	Robinson *et al.*	1945, *Am. J. Physiol.*, **143**: 21.
Thermal Acceptance Ratio [1]	OQMG (U.S.)	1945, *U. S. Army Off. Quartermaster Gen.*, Aug. 20 and Sept. 17.
Index of Environmental Warmth	Bedford	1946, *Brit. Med. Res. Counc. War Memo*, 17.
P4SR (Probable 4-Hour Sweat Rate)	McArdle *et al.* Queen Square	1947, *Brit. Med. Res. Counc.*, R.N.P. 47/391.
HSI (Heat Stress Index) [1]	Belding, Hatch	1955, *ASHAE J. Sect., Heat. Piping Air Cond.*, Aug., 129.
WBGT (Wet Bulb, Globe Temperature)	Yaglou	1957, See Minard *et al.*, *JAMA*, **165**: 1813.

[1] Attempt at quantitative expression of heat load.

QUANTITATIVE APPROACH VS. EMPIRICAL

Some of the approaches to the problem have been empirical. We argue that the number of variables to be considered foredooms development of a simple, comprehensive heat index on that basis. The more promising approach, and the one which is also more challenging and intriguing, seems to us to be the quantitative one. It has been used by a number of investigators, beginning with Winslow, Herrington and Gagge at the Pierce Laboratory in the early 1930s. This approach is consistent with that established for hygienic practice in industry and public health generally: identify the noxious factors and quantify them as basis for remedial action.

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[1] Stress is affected by temperature of air, solid surroundings and skin, ambient and skin vapor pressures, air speed, body surface area and postural attitude, body heat production, clothing, and time of exposure. Exposure involves strains of production of sweat, of circulation of blood to the skin and of elevation of body temperature.

DESCRIPTION OF HEAT STRESS INDEX (HSI) OF BELDING AND HATCH

Prof. Hatch and I have proposed a relatively simple index based on knowledge of air and wall temperatures, ambient water vapor pressure, air speed and grade of activity. The coefficients established at Fort Knox are utilized to compute heat flow by Radiation (R) and Convection (C). To these we add the man's own heat production (M, for metabolism) to establish the heat load:

$$M+R+C=\text{healt load}$$

This is the load which must be dissipated if constant temperature of the body core is to be maintained. The available avenue for dissipation of whatever heat load there may be is evaporation of sweat. So

$$\text{heat load} = \text{evaporation required for heat balance} = E_{req}$$

It has been our thesis that the strains of exposure are proportional to the demand for sweat and that uncompensatable heat stress (i.e., overstress) is encountered whenever the heat load exceeds (*a*) the ability to sweat, or (*b*) the ambient capacity for evaporation of sweat (E_{max}). Several investigators have established that the capacity to sweat over a period of hours is about a liter an hour. Evaporation of a liter can remove about 600 KCal. of heat. The capacity for evaporation is dependent on the difference in vapor pressure between the skin and air, and on wind speed. The coefficient applicable for nude man has also been provided by the Fort Knox studies. We have proposed that heat stress in terms of percent taxation of human capacity to maintain heat balance, is represented by

$$\frac{E_{req}}{E_{max}\ (\text{up to }600)} \times 100$$

where all value are in kilocalories. This we have called the Heat Stress Index (HSI). For simplicity of computation of heat exchanges by the various avenues, skin temperature was assumed to be 35°C, regardless of the exposure conditions.

CAPABILITIES AND DEFICIENCIES OF HSI

We and others have now had considerable experience with this system for grading heat stress. Under many circumstances the reported heat strains have correlated well with those predicted by the index values. Under other circumstances, particularly under some conditions predicted to be critically hot, the index has proven less than satisfactory.

Principal problems which have arisen are relatable to (*a*) the use of a fixed skin temperature for computation of E_{req} and E_{max}; (*b*) the assumption that circulatory strain involved in bringing heat to the skin is proportional to the strain of moving the heat from the surface of the skin to the surrounding air, and (*c*) deficiency of understanding of the effects of clothing. We shall cite an example illustrative of each of these problems.

a. MISESTIMATE OF HEAT STRESS ARISING FROM ASSUMPTION $T_s = 35°C$.

One of our principal interests, since we are part of a Department of Occupational Health, has been in jobs involving intermittent exposure to high levels of heat with a large component of radiation. The objective in this particular instance was to set up a job which would result in a significant rise in body temperature and to study effects of recovery conditions on the rate of unstorage of this heat.

A situation was created which, on the assumption of $T_s = 35°C$, represented

$$\frac{M+R+C}{E_{max}} = \frac{280+290+230}{330} = \frac{800}{330}$$

all values in KCal./hour. During the exposure we wanted the subject to store about 100 KCal. of heat in the body, represented by a rise in average body temperature (core and skin) of 1.7°C. We predicted that he would store at the rate of 800 — 330=470 KCal./hour. His predicted exposure time for storage of 100 KCal. was 13 minutes.

In actual fact, the subjects under these conditions exhibited skin temperatures which rose rapidly to about 37.8°C, then leveled off. This meant that the gradient for heat gain by R and C was actually reduced by 2.8°C and the effective vapor pressure gradient was raised by 7 mm Hg. Revised prediction of heat load and E_{max} on this basis gave

$$\frac{M+R+C}{E_{max}} = \frac{280+260+190}{470} = \frac{730}{470}$$

The underestimate of T_s in the original prediction meant an 11 % overestimate of the heat load and, much more important, a 30 % underestimate of E_{max}. The revised estimate of time required for storage of 100 KCal. is 75 % longer, 23 minutes.

Three subjects actually endured 19 different exposures to these conditions for 30 minutes, with an observed storage of about 100 KCal. and apparent evaporative cooling (from weight loss) of 295 KCal. Thus the actual heat load appears to have been $(295 + 100) \times 2 = 720$ KCal./hour, and the actual evaporation to have been at the rate of 590 KCal./hour. (The evaporation estimate may be high because some of the sweat may have dripped off.)

This example has been cited to illustrate the point that arbitrary assumption of $T_s = 35° C$ can give rise to serious errors in estimating evaporative potential, and thereby yield overestimate of strain of exposure and underestimate of tolerance time[1].

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[1] The example also illustrates a finding for which there is much additional support, that observed heat loads and evaporation rates for semi-nude men corre-

b. FAILURE OF INDEX TO REFLECT CIRCULATORY STRAIN.

Anyone who has been observing physiologic responses will grant that under many circumstances circulatory strain is the critical factor. Yet attempts to rate heat stress have largely ignored this (Robinson's being the conspicuous exception) in favor of sweating and body temperature rise. This may be because heart rate is the only index of circulatory strain that is convenient to measure, and heart rate per se has been maligned as an index of overall circulatory demand. Our attention has been called to this subject in cases of incipient heat exhaustion. The typical picture, in our laboratory, is a rapidly rising pulse rate. We now recognize that a sitting subject may exhibit this runaway pulse after his heart rate reaches 100 to 110 beats per minute, that during light work while standing the critical rate may be 130 beats per minute, while with hard work the rate may be 160 or higher. Presumably the difference in rates at which failure arises is related to the role of activity in promoting venous return (milking action on veins).

The basic relation of circulatory strain which we overlooked in developing our index was the fact that the amount of heat to be carried to the skin by the circulation depends entirely on M. The strain of carrying it depends on the gradient between core (T_r) and skin temperatures. The circulatory demand is approximately represented by $\frac{M}{T_r - T_s}$ [1]. The ability to meet the demand varies with the activity. Robinson's data, and our own, suggest that when sitting the maximum prolonged blood flow to the skin is about 1.4 l per minute (calculated on this basis), whereas when walking at a good pace the capacity may rise to 2.8 l or 3.0 l per minute.

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spond well with predictions derived with the Fort Knox coefficients. The coefficients should be accepted as approximately correct.

[1] A more precise expression of circulatory demand requires consideration of the fixed resistance to heat flow represented by the layer of skin between the capillaries and the surface. Kerslake (*J. Physiol.*, **127**: 280, 1955) and Hatch (unpublished) have provided means for estimating this resistance.

HSI can fail to reflect the circulatory burden. As illustration, we take two situations with different metabolic rates, but similar heat loads, with $T_s = 37°C$ and $T_r = 38°C$.

	M		R + C		Heat load
sit	90 KCal./hr.	+	260	=	350
walk	350	+	0	=	350

Let us assume that E_{max} is also 350 KCal./hour (HSI = 100). With this one degree gradient of skin temperature, the required circulation to the skin while sitting is 1.4 l per minute, and, as indicated in the preceding paragraph, can be met. While walking the required circulation is 5.7 l per minute; this cannot be met if the maximum capacity is 3.0 l. Thus we have two situations with equivalent Heat Stress Index values, and entirely different orders of circulatory strain. If the walking situation were to be tolerated by the working man, it would have to be at the cost of a further 1°C elevation of core temperature, which is in itself a manifestation of strain. It is obvious that a comprehensive Heat Stress Index should take into account the circulatory strain referable to bringing metabolic heat to the skin.

c. COMPLEX EFFECTS OF CLOTHING.

Space does not permit detailed treatment of the complex effects of clothing on heat stress and strain. Certain of the empirical indices provide adjustments or separate scales to differentiate between the nude man and one who is clothed in ordinary shirt and trousers, or overalls. Within the ranges covered, the wearing of clothing has generally been represented as a burden, as a factor increasing strain. That this is not always so has been demonstrated under desert conditions, when air and/or radiant temperature exceeds skin temperature. To be provocative, an experience is cited which once again suggests caution in arbitrary acceptance of our own index values.

We had occasion to referee a question of overly stressful exposure at an industrial job which involved working between two furnaces. Each man was required to work fairly hard for about four minutes, then was permitted to rest for two minutes in a draft of out-of-door air. The temperature of this air reached 35°C or more in summertime. The six-minute work-rest cycles were repeated for two two-hour periods during each eight hour shift.

On the spot measurements indicated that the average hourly heat load of this job, combining work and rest periods, could reach 630 KCal. per hour. Production and evaporation of a little more than 1 l of sweat per hour would be required to maintain thermal equilibrium. Since this job required only four hours of exposure per shift, the sweating requirement could not be regarded as excessive. However, part of the assessment of any hot job is the determination of E_{max}, to see whether ambient vapor pressure and air speed permit evaporation of the sweat that is required. Available data indicated that vapor pressure might rise to 19 or even 22 mm Hg in summertime. We therefore computed E_{max} for $T_s = 35°C$. At 17 mm Hg this was 530 KCal./hour; at 22 mm it was 430. In view of the load of 630 KCal. it was concluded that the job was excessively stressful. However, despite worker complaints, there was no history of heat injury on this job, and in an effort to learn why, we simulated the job in our laboratory.

Subjects alternately worked and rested in accordance with the six-minute cycle, for two hours. They were dressed in the clothing most commonly worn on the real job, cotton khaki shirt and trousers, quarter-sleeve cotton under-shirt and full length underdrawers. Since ambient vapor pressure appeared to be the critical factor, this was controlled, at 17, 19, 22, 25 and 27 mm Hg[1]. The studies revealed that the actual heat load was only 70 % of what had been predicted. (Here we used the actual skin temperature in the calculations.) The explanation must be that the clothing was blocking some of the radiation. With this revised estimate of heat load, evaporation should not limit heat balance until vapor pressure reached about 22 mm Hg. The data on strains of these exposures bore out this prediction. Sweating, rectal temperature, recovery heart rate, metabolic heat production and fatigue were stable at vapor pressures between 17 mm and 22 mm, but rose at 25 and 27 mm Hg.

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[1] Subjects alternately worked, pulling against a 30 pound load 6 times a minute at $T_g = 61°C$, $T_a = 39°C$, $V = 76$ m/min., and sat quietly at $T_g = T_a = 35°C$, $V = 120$ m/min.

Heat balance was nearly maintained even at 27 mm Hg, but at cost of much extra sweat production. We suggest that this was accomplished by soaking the clothing, which results in presentation of much extra wetted surface. Evaporation from clothing is less efficient for skin cooling; this is suggested by the apparent utilization of 530 KCal. of sweat to meet a 430 KCal. heat load.

The postulation that the clothing reduced the heat load of this situation requires support in view of the possibility that the basic heat transfer coefficients for R and C are wrong. This point was examined for the present situation by exposing the same subjects both semi-nude and clothed. The radiation load was reduced by about 1/3, to a level tolerated with bare skin[1]. Predicted load due to Radiation and Convection was now 330 KCal./hour. The load computed from the caloric value of the sweat actually evaporated, with a slight correction for storage of heat by the skin, was within 10 % of that predicted for the semi-nude man, which is considered good agreement. For the clothed man it was 30 % less than for the semi-nude. This is explainable on the basis that radiant energy absorbed by the clothing is dissipated only partially to the skin, the remainder being lost by convection and re-radiation to the environment.

Thus, clothing may importantly reduce heat stress by blocking R and C. It may reduce evaporation from the skin, but this may be compensated partially or fully by increased sweat production which wets the clothing, giving a larger total surface for evaporation.

CONCLUSIONS

1. With the large number and complex relationships of factors that are involved, the construction of a universally valid heat stress index must rest on the quantitative approach for: (*a*) assessment of heat load; (*b*) assessment of potential for moving metabolic heat from its source to the skin, via the circulation; (*c*) assessment of

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[1] At work, $T_g = 52°C$, $T_a = 36°C$, V.P. = 22 mm Hg, V = 76 m/min.; at rest, $T_g = T_a = 35°C$, V.P. = 22 mm Hg, V = 120 m/min.

the potential for dissipation to the environment by evaporation; and (*d*) assessment of tolerance time in the presence of overstress. Currently available knowledge is believed nearly adequate to permit construction of such a heat stress index.

2. Four principal problems of this approach are: (*a*) how properly to predict skin temperature and make use of it in computing E_{max}; (*b*) how to predict circulatory strain and take it into account; (*c*) how to predict effects of clothing on R and C and on E_{max}; and (*d*) how to predict tolerance time when stress is excessive.

3. Several of the indices that have been produced are valid and useful in the ranges of conditions for which they were proved. The recommendation for those who are not specialists in this field is to select a scale which was proven under conditions similar to your own.

THE PHYSIOLOGICAL STATE AT THE LIMIT OF TOLERANCE

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The principal papers in this section have dealt with the practical problem of characterizing the environments which a man can tolerate with comfort or without loss of efficiency during a working day. By way of discussion I should like to consider conditions that can be tolerated only for shorter periods and to review the physiological basis for the sensation that the environment has become intolerably hot. Two sets of data will be presented. One set was obtained from 6 men walking on a treadmill while wearing various assemblies of protective clothing at the U. S. Army Chemical Center (1, 2, 3). The other was obtained by Dr. Taylor and his colleagues in California during exposures of 12 men at rest to very high temperatures (4, 5, 6). In addition to their published reports, I am very much indebted to Dr. Taylor's group for some of their unpublished data which were needed to complete our analysis of the problem.

The common feature of these two experiments was that the subject terminated each exposure when he had a generalized sensation of being unbearably hot. The physiological observations made at or immediately before this end point are plotted according to the duration of the exposure, called the voluntary tolerance time. The range of tolerance times was produced in our experiment mainly by varying the resistance of the clothing to evaporative heat loss and in Dr. Taylor's experiment by varying the environmental temperature.

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Fig. 1 shows the total sweat production during the exposure in terms of nude weight loss. The open circles refer to the California data; the solid circles to ours. The slopes and the 95 % confidence limits of the slopes are given together with the standard deviation of each observation. As would be expected, the sweat production increased with the tolerance time; however, the total was less than the 4.5 l, which is the basis of the predicted 4 hours sweat rate index of McArdle *et al.* (7).

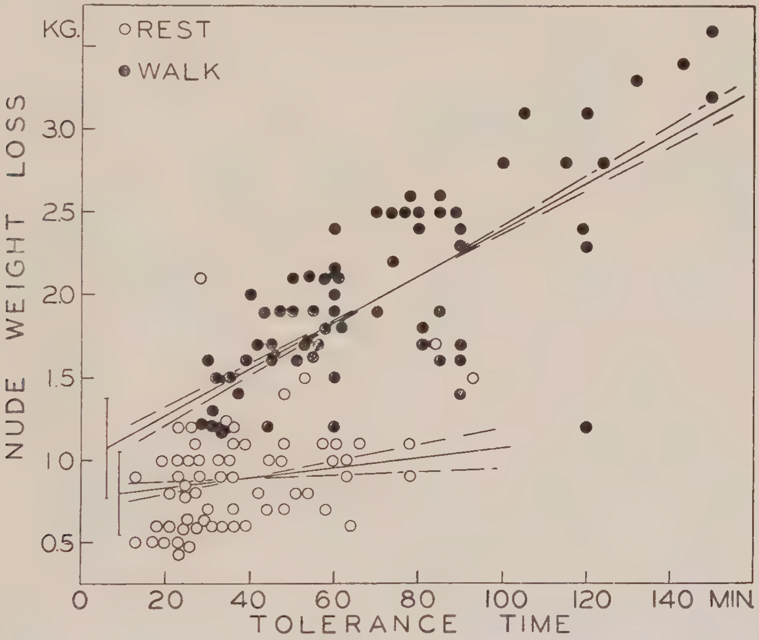

Fig. 1

Fig. 2 shows the final heart rate recorded during the period of exposure. In both sets of data, the heart rate declined as the tolerance time increased. Particularly with the longer tolerance times, it is clear that the heart rates at termination are well below those found in severe exercise.

Fig. 3 shows the final skin temperature. This is the mean of the temperatures of thermocouples located at representative points on the body. The final skin temperature also declined as the tolerance time increased. In the resting set of data the air temperatures ranged from 60°C to 117°C, but the subjects were protected from painful contacts.

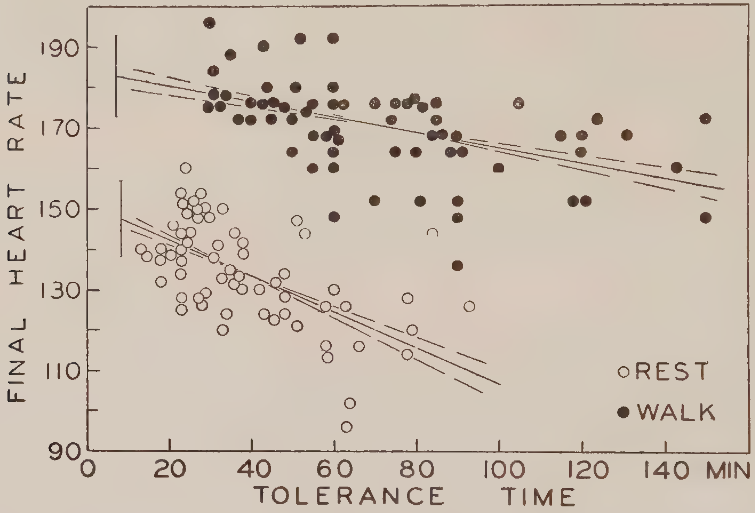

Fig. 2

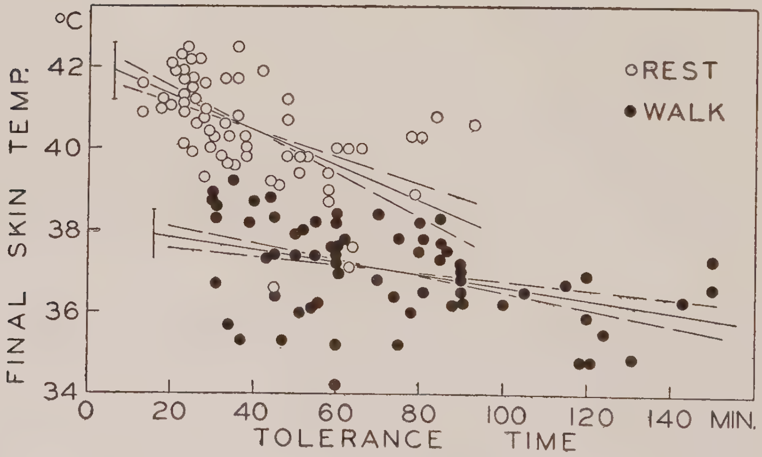

Fig. 3

Fig. 4 shows the final rectal temperature. In the walking tests this did not change significantly from the short to the long tolerance times. If we were considering only this set of data, we would accept the final rectal temperature as the physiological measurement most closely associated with the decision to terminate the exposure. However, in the resting experiments, the final rectal temperature increased significantly with the tolerance times, so that this index is probably not generally related to the decision to terminate.

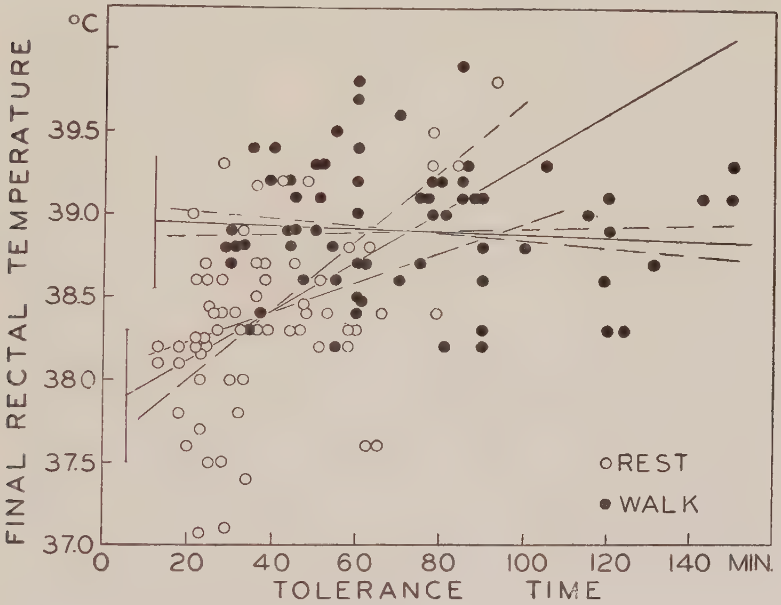

Fig. 4

Fig. 5 shows the increase in the mean body temperature from the beginning to the end of the exposure. In arriving at the mean the skin temperature was weighted one-third and the rectal, two thirds. In neither set of data was the slope significantly different from zero. Hence, it is concluded that, of the indices considered, the increase in mean body temperature was the one most closely correlated with the decision to terminate the exposure. The data are somewhat equivocal for the means for the two conditions are quite different. There is the possibility that the critical rise is not the same in rest and exercise. However, there were wide man-to-man diferences within the two groups of subjects so that I prefer to attribute the difference between the two experiments to the men rather than to the conditions. It would be most desirable to repeat this work with the same men exposed to a greater variety of conditions.

As the data stand, however, it would appear in the case of a single man that the decision to terminate the exposure was reached at the time the mean body temperature had increased by an amount independent of the duration of the exposure. The usefulness of this observation arises from the fact that the rise in mean body temperature is a measure of the heat accumulated in the body. The relationship between voluntary tolerance time and rate of heat storage provides a way of entering the heat balance equation with a performance term (8, 9). Hence, one has the theoretical basis to calculate the tolerance time from the other terms of the heat balance equation as Blockley *et. al.* (10) have done. Belding and Hatch (11) have used essentially the same relationship in their eighth equation to take care of situations in which the requirement for heat loss by sweating is greater than the heat acceptance of the environment.

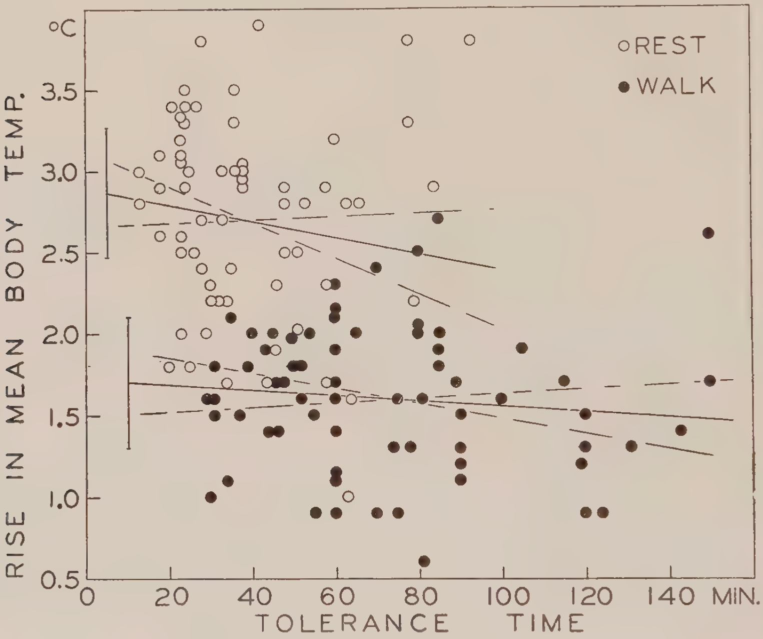

Fig. 5

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INDUSTRIAL HEAT STRESS

FREDERIK HENDRIK BONJER

Men in industry are supposed to do work. This means that the metabolism often will be higher than is encountered in studies of acclimatization to tropical zones. On the other hand, exposure to heat will be restricted to only one third of each period of 24 hours. Thus industry may create special problems resulting from the combination of work load and heat load.

Heat stress may be imposed on the man either by radiation or by high humidity in addition to high air temperatures. The environment may not only create an abnormally high heat gain, but can also interfere with the mechanism for heat loss. In this way intolerable conditions can be reached.

As a principle both long term and short term effects could be expected. There is not much known however about pure long term effects of exposure to industrial heat. According to some authors, atherosclerosis or nephrolithiasis could be expected. Our observations did not show any evidence however and we were not able to prove any reliable difference in physical fitness between industrial workers with and without heat stress. This does not demonstrate that there are no long term effects, but they are not easily found. Natural selection amongst the workers might play a role.

Much more is known about short term effects of industrial heat stress. Studies were made on the physiological responses during and immediately after exposure and many authors developped heat stress indices to indicate the degree of strain on the worker.

It is not always enough to know wether there is an industrial heat stress or not and, if so, to what extent. Managers want to know the allowable working time for each job and the required period of rest after each exposure.

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This problem may be approached by the use of a heat balance as indicated by Hatch, Haines and Belding. Changes in the heat content of the body (H) can be computed from the heat production by metabolism (M) and the rate of heat exchange by radiation (R), convection (C), conduction (D) and the rate of heat loss by evaporation (E):

$$\Delta H = M + R + C + D + E$$

It will be understood that M is always positive and E is almost always negative. R, C and D, however, may be positive or negative according to the situation. If the algebraic sum equals zero, there is no change in the body heat content H. This is by far the most common condition of the body. The influences of muscular activity and from the environment are compensated by the thermoregulation of the body and mean body temperature is kept constant at a certain level. This level is not duely 37°C, but is dependent from the type and intensity of the work done and from the environmental factors.

Unbalance will occur only if the regulatory mechanisms are failing. Heat gain by excessive radiation combined with intense muscular activity for instance may exceed the maximal heat loss by evaporation. In such a case the algebraic sum will be positive and ΔH will get a positive value. The heat content of the body will increase as does the mean body temperature.

The essential idea of our conception of heat stress evaluation is the assumption that this rise in mean body temperature should never exceed the value of 1°C. As the rate of heat gain of the body (ΔH) follows from the equation for the heat balance, the maximal allowable time of exposure may be calculated.

An example may give the opportunity to discuss the advantages and weaknesses of the presented method.

A man with a body weight of 67 kg and a total surface area of 1.85 m^2 is unloading a brick furnace at a metabolic rate of 6.35 kilocalories per minute, 5 % of which is not converted into heat, but into mechanical work. The loss of body weight is 13.5 g per minute.

The mean temperature of the skin (t_s) is 35°C. The dry bulb temperature (t_a) is 41.6°C, the globe temperature (t_g) 57.6°C and the air velocity 75 cm per second. The relative humidity is 15 %.

Before we use these data to compute the allowable working time in the furnace we may discuss their validity.

The measurement of body weight and its smaller changes requires a reliable balance with an accuracy up to 5 g. The surface area is calculated from the Du Bois tables using data about sex, age, body weight and length.

The metabolic rate can be calculated from the oxygen consumption per minute and the respiratory quotient, obtained either by Douglas-bag or by any good portable gasmeter. To find the amount of produced heat (M) the mechanical work expressed in caloric units should be subtracted from the calculated metabolism. In the laboratory the mechanical work can be measured on an ergometer, but in the field we often should make a guess. The error is to be neglected however.

The loss of body weight should indicate the amount of water evaporated from the skin and lungs. An error is made if sweat drips from the skin as this causes weight loss without heat loss. Mean skin temperature is difficult to be measured as such. An approximation is derived from the skin temperature on various sports. No attention will be given in this paper to the methods of climate measurement. It should be stated however that the mean radiant temperature (t_r) is derived from the temperature of the globe according to Vernon, the air temperature and the air velocity.

The next step of the procedure is the calculation of heat exchange by various mechanisms from the climatological data.

The heat exchange by radiation may be computed by a modified formula according to Stefan-Boltzmann:

$$R = A_r \cdot K_r (t_r - t_s)$$

in which K_r is supposed to be a constant. A_r is a percentage of the total body surface according to the working posture. In our example

$$A_r = 0.77 \times 1.85 = 1.43 \text{ m}^2$$
$$K_r = 0.115$$
$$t_r = 73^\circ\text{C}$$
$$t_s = 35^\circ\text{C}$$
$$R = 1.43 \times 0.115 (73\text{-}35) = 6.3 \text{ kcal./min.}$$

The heat exchange by convection can be computed by the empirical formula of Winslow, Herrington and Gagge:

$$C = k \cdot A_c \cdot \sqrt{V} . (t_a - t_s)$$

For k we took in our example 0.8 kcal/m²/h and for A_c, the effective convective area, 97 % of the total body surface.

$$k = \frac{0.8}{60} = 0.013$$
$$A_c = 0.97 \times 1.85 = 180 \text{ m}^2$$
$$V = 57 \text{ cm/sec.}$$
$$t_a = 41.6^\circ\text{C}$$
$$t_s = 35^\circ\text{C}$$
$$C = 0.013 \cdot 1.80 \cdot \sqrt{57} \cdot (41.6 - 35) = 1.0 \text{ kcal./min.}$$

The heat exchange by conduction (D) can be disregarded if there is at most a minimal contact of the skin with the surroundings. The conditions under which the measurements in this study were carried out justified such disregard.

The heat loss by evaporation (E) may be assessed from the product of the weight loss per minute and the heat of evaporation of 1 g of water (0.58 kcal.).

$$E = \text{weight loss/min.} \times 0.58$$

In our example the weight loss of 13.5 g per minute gives:

$$E = 13.5 \times 0.58 = 7.8 \text{ kcal./min.}$$

This figure gives the actual heat loss by evaporation. It might be possible however that the maximum evaporative capacity is much more. In the assessment of the maximum allowable working time this maximum evaporative capacity has to be taken into account. Haines and Hatch provide a formula in which this capacity can be estimated.

$$E_{max} = 3.33 \cdot V^{0.37} \cdot (P_s - P_a) \text{ kcal./h}$$

where

V = air velocity in cm/sec.
P_s = water vapor pressure of the sweat on the skin in mm Hg.
P_a = water vapor pressure in the air in mm Hg.

In our example

$$V = 15$$
$$P_s = 42$$
$$P_a = 9$$

$$E_{max/min} = \frac{3.33}{60} \cdot 4.48 \cdot (42 - 9) = 8.2 \text{ kcal./min.}$$

It follows that in our example the actual heat loss by evaporation (7.8 kcal./min.) nearly equals the maximum.

When we substitute the values from the example in the equation:

$$\Delta H = M + R + C + D + E$$

we find for ΔH $(6.35 - 0.05 \times 6.35) + 6.3 + 1.0 + 0 - 8.2 =$
$= 5.1$ kcal./min.

The man in our example had a body weight of 67 kg. At an increase of his mean body temperature of one degree centigrade and a specific warmth of 0.83 a heat gain of $67 \times 0.83 = 55.6$ kcal. would be allowable.

As ΔH has a value of 5.1 kcal./min., the allowable time of exposure would be $\frac{55.6}{5.1} = 11$ minutes.

The formulae for the assessment of heat exchange by radiation and convection as proposed by Hatch and Haines were slightly modified and used in a field experiment. This was described by Snellen, who could show a good accordance between the calculated value ΔH and the directly measured increase in body temperature.

This, of course, does not prove the over all usefullness of the formulae mentioned above. Research should be done in the future on a better assessment of the effective body areas, both for radiation and convection, at various working postures.

The influence of clothing was neglected.

Another practical problem is the assessment of the required recovery time after each exposure. If rest is taken in a moderate climate, the heat content of the body may decrease linearly or in an exponential way.

If it would be a linear process, we could use the same technique for the determination of the recovery time from the rate of heat loss (ΔH).

It is more likely, however, that the decrease in heat content is an exponential function. As long there is no more knowledge about recovery processes we will have to manage with symptoms as pulse rate and rectal temperature in order to indicate the minimum required recovery time under industrial conditions.

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THE ASSESSMENT OF INTOLERABLY HOT ENVIRONMENTS FOR WORKING MEN

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When men work in surroundings so hot that they cannot establish bodily thermal equilibrium, the conditions of exposure are often too severe to fall within the range of use of existing heat stress indices. Thus, when the tolerance of human subjects to such excessively hot conditions is considered, there is often no method available for comparing the severity of different climates. My colleagues Dr. J. S. Weiner, Dr. R. F. Hellon and myself were faced with this problem after we had described the «tolerance times» of men working hard in various saturated environments (Lind, Hellon, Weiner & Jones, 1955) and then had to extend these findings to embrace a wide range of non-saturated environments. In order to reduce the experimental programme to manageable dimensions, it was clearly necessary to apply some method by which the severity of different climates could be compared and assessed. The combination of work and climate we wished to investigate resulted in a total heat stress too severe to fall within the range of use of all recognised indices, except for the Effective Temperature Scale (Houghten & Yaglou, 1923), the accuracy of which has often been criticised at such high levels of heat stress, both for working men (e.g. Robinson, Turrell & Gerking, 1945), and for sitting men (Lind & Hellon, 1957).

A simple formula was derived from previous work (Ellis, Ferres, Lind & Newling, 1953) whereby the dry and wet bulb temperatures could be weighted to relate climates of similar severity, as follows, WD (weighted temperatures equivalent) = 0.15d (dry bulb temperature) +

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$+ 0.85w$ (wet bulb temperature). This formula was considered possible as a realistic method of comparing the severity of excessively hot environments. No allowance was made for air movement, because the proposed air movement in the present experiments was similar to that used by Ellis *et al.* (1953) and these experiments were designed to meet a specific requirement in conditions where the air movement tends to remain low.

METHODS

Sixteen of the original volunteers used in the previous experiments in saturated environments (Lind *et al.*, 1955) acted as subjects. The experimental procedure was exactly the same as was used in those experiments, and the subjects were exposed to 4 non-saturated environments, (1) 100/88 (37.8/31.1), (2) 120/79 (48.9/26.1), (3) 120/85 (48.9/29.4) and (4) 120/94 (48.9/34.5) in °F (°C). Air movement was 175 ft/min. in all these experiments. The average energy cost of the standard work routine was 200 kcal./m^2/hr. Shorts, vest and boots were worn. The subjects were divided into two groups, each of which wore one of two types of self-contained breathing apparatus commonly worn underground in mines-rescue operations, when noxious gases are often present. An interval of three weeks was allowed between successive exposures for each subject, to minimise acclimatization effects.

RESULTS

The table shows the average «tolerance times» of the 16 subjects in each of the 4 climates investigated. Compared with these times are the predictions calculated from a knowledge of the «tolerance times» found in saturated environments in the previous experiments, and using the «WD» formula. None of the predicted values varied by more than 5 minutes from the recorded durations of exposure. Similar predictions based on the Effective Temperature Scale varied from the actual durations by 6-13 minutes.

The average observed «tolerance times» with ranges shown in brackets for 16 men exposed to 4 environments, compared with the predicted durations of «tolerance» using the «WD» formula as a basis for prediction

Environments		Observed tolerance time		Predicted tolerance time	
D.B. (°C)	W.B. (°C)	Apparatus		Apparatus	
		1	2	1	2
48.9	26.1	55 (47–65)	62 (54–65)	60 (52–65)	63 (56–65)
37.8	31.1	56 (54–65)	63 (59–65)	54 (49–54)	62 (54–65)
48.9	29.4	55 (52–59)	61 (49–65)	53 (49–54)	62 (54–65)
48.9	34.5	43 (49–48)	46 (41–49)	39 (35–44)	42 (39–46)

DISCUSSION

The simple weighting of dry and wet bulb temperatures adequately described environments where the thermal stress was similar for working men, in terms of «tolerance times». The rate of work, the amount and type of clothing, and the air movement remained constant in the present experiments. The use of such a formula was first considered likely to apply only to a very restricted range of conditions of exposure to heat. But comparison of the present results with those of Eichna, Ashe, Bean & Shelley (1945), Robinson *et al.* (1945) and Ellis *et al.* (1953) showed that climates of comparable severity were similarly related by a simple weighting of dry and wet bulb temperatures in all these different series of experiments, despite differences in the criterion of estimating «tolerance», wide differences in the energy expenditure and in the amount of clothing worn and some variation in the air movement.

In these different series of experiments energy cost varied between 110-200 kcal./m^2/hr. clothing from the nude to the fully clothed state, while the air movement varied between 150-265 ft/min. The weighting of dry wet bulb temperatures which expressed environments of similar severity ranged from $0.1d + 0.9w$ to $0.15d + 0.85w$.

It seems that, for practical purposes, a simple formula of this type may be of some value in assessing and comparing the severity of excessively hot climates, provided that they do not vary widely from the conditions which existed in the different series of experiments mentioned above. A knowledge of the tolerance of men to given conditions of exposure to a small number of excessively hot climates will allow reasonably accurate prediction of the tolerance to a much wider range of climates.

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HUMIDITY AND HEAT STRESS

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Physical aspects of the heat stress situation have been examined by Machle and Hatch (1947), Brunt (1947) and Woodcock, Powers and Breckenridge (1956). Although there are differences in the approaches used by these authors, their conclusions about the effect of humidity on heat stress, when other parameters are kept constant, are similar. Two fundamentally different situations must be distinguished; one in which all the sweat produced is evaporated at the skin surface, and the other in which the rate of sweat production exceeds the rate of evaporation, the latter depending only on the vapour pressure gradient from wet skin to air and on the physical characteristics of the skin/air interface.

When the sweat is evaporating at the same rate as it is produced, the rate of production is adjusted physiologically to the needs of the situation, heat balance being maintained when the body has become warm enough to initiate the appropriate sweat rate. Variations in ambient humidity have a negligible effect on heat transfer by convection, but do cause small changes in the heat lost from the respiratory tract and by diffusion of water through the skin. The rate of sensible sweat production therefore has to increase slightly with rising humidity, but only enough to maintain the total rate of evaporation from the body.

When the rate of evaporation is limited by physical conditions at the skin/air interface, the skin temperature in the steady state can be predicted from physical considerations and a knowledge of the rate of heat production. Deep temperature will be driven to such a level that the transfer of heat to the skin can take place fast enough

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to prevent any further rise in deep temperature. Sweat rate will be without effect on heat exchange, but will increase greatly as the skin and deep temperatures rise with increasing ambient humidity.

If, therefore, one considers a situation in which air temperature, wall temperature, air movement and metabolic rate are all held constant, but in which ambient humidity is allowed to vary over a wide range, one would expect to find that at low humidities changes in humidity would have little effect on skin and deep temperatures and sweat rate, whereas above a certain critical humidity (at which the skin becomes completely wet with sweat) changes in humidity would cause large changes in these quantities.

Experiments have been performed on nude resting men to check this expectation. The results for one subject are presented in Fig. 1.

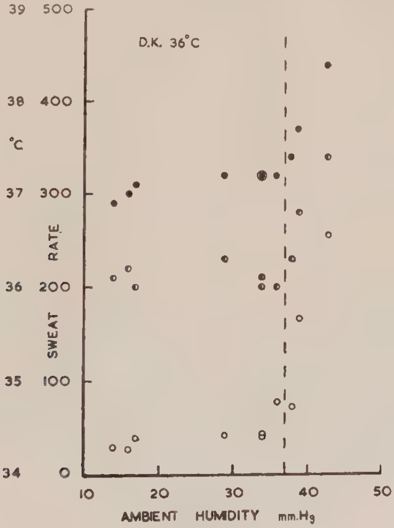

Fig. 1

The observations are means for the period 40-60 min. after entry into the test environment. They show the expected inflexion at a critical ambient humidity, and calculation suggests that the skin would reach complete wetness at the humidity indicated by the vertical dotted line. The transition between the two states appears to be abrupt. Similar results have been obtained on another subject and at another temperature (Brebner, Kerslake & Waddell, 1958).

It is concluded that one feature of any satisfatory system for the expression of heat stress must be that the effect of humidity on heat stress is discontinuous, being small below the critical humidity and very large above this level.

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GENERAL DISCUSSION

Dr. A. R. Lind (U.K.) — With reference to the technique described by Dr. Craig, we have similarly calculated heat storage in our own data as well as in those of Dr. Craig's and Dr. C. L. Taylor's. Calculated heat storage, in Kcal./m^2, plotted against tolerance time appeared to show a smooth, parabolic relationship. Statistical analysis, however (for which I am indebted to Mr. F. D. K. Liddell), showed major differences to exist between results obtained in different series of experiments, and apart from the expected differences due to different subjects there were strong indications that a large proportion of the remaining variation derived from the inaccuracies of the formula used for calculating heat storage. This is, perhaps, not surprising considering the wide range of conditions in all the experimental series we have examined. Our conclusion was that this method could not be applied to the estimation of tolerance times unless a more accurate method of calculating heat storage becomes available.

Dr. S. Robinson (U.S.A.) — Has Dr. Belding taken acclimatization of men to heat into account in formulating his index?

I would like to ask Dr. Craig if he has studied, in men exposed to extreme heat, the temperature gradient between rectal and mean skin temperature of the men in relation to the end point of heat tolerance.

Dr. F. Craig (U.S.A.) — In reply to Dr. Robinson's question, the difference between skin and rectal temperature has not been analyzed in relation to the tolerance time, although the data are available. However, the heat storage has the merit of beeing related at least theoretically to the other terms in the heat balance equation.

Dr. D. E. Bass (U.S.A.) -- Do the indices have any allowance for dehydration as a limiting factor in performance? Adolph has shown that 5 % dehydration leads to decreased performance.

Dr. E. M. Glaser (U.K.) — I am wondering what allowance can be made in the construction of thermal indices for the existence of individual variations. There are not only variations among similar individuals of similar age, but there are also great differences between people under 45 and older people, as shown by Dr. Lind and his colleagues in the *Journal of Physiology* in 1957.

Dr. G. P. Crowden (U.K.) — Referred to the sweat efficiency values of 100 % for nude and 67 % for clothed subjects, quoted by Dr. Belding. He asked whether this agreed with the recommendation of Dr. C. P. Yaglou in 1925, that workers in the heat should wear singlet vests, which retained sweat which would otherwise drip off their bodies. He wondered whether the retention of this sweat in the vest enabled greater evaporative cooling of the body to be attained.

Dr. S. Robinson (U.S.A.) — We have found that when men who are fully acclimatized to the heat are resting in a cool environment, their blood volumes are not expanded above the usual control values, but when the men work in the heat, their circulating blood volumes increase within 3 hours by an average of 15 % above the control values. This expansion of the circulating blood volume does not involve a change in hematocrit from the control.

Dr. F. Craig (U.S.A.) — In reply to Dr. Lind's remarks, I do not defend the present technique of measuring heat storage. Perhaps better data will be produced with Commander Minards apparatus.

3RD SESSION: **Heat ilness**

Wednesday, September 10th — 3:00 to 4:30 p. m. Room A

The meeting was held under the chairmanship of Dr. Jean Sénécal.

The Rapporteur was Dr. C. S. Leithead and the Secretary was Dr. Carlos F. de Oliveira.

The following papers were presented and discussed.

NOMENCLATURE AND CLASSIFICATION OF THE HEAT DISORDERS

W. P. STAMM

I have been asked to speak on the classification of heat disorders in order to stimulate discussion of this subject in the hope of reaching sufficient agreement to produce a classification suitable for inclusion in the new edition of the *International Statistical Classification of Diseases, Injuries, and Causes of Death* due in 1967.

First I think we should be quite clear about the functions of a statistical classification and the distinction between it and a nomenclature.

A nomenclature attempts to assign a name to every clinical or pathological condition which can be specifically identified, however difficult or intricate the diagnostic procedure may be.

To quote from the introduction to the *Manual of International Classification of Disease,* «the purpose of a statistical compilation of disease data is primarily to furnish quantitative data that will answer questions about groups of cases... A specific disease entity should have a separate title in the classification only when its separation is warranted because the frequency of its occurrence, or its importance as a morbid condition, justifies its isolation as a separate category... the doctors who practise and who will be making entries in medical records or writing medical certificates of death were educated at different medical schools and over a period of more than fifty years. As a result, the medical entries on sickness records, hospital records, and death certificates are certain to be of mixed terminology which cannot be modernised or standardised by the wave of a magician's wand. All these terms, good and bad, must be provided for as inclusions in a statistical classification».

It follows from this that the idea apparently held in some quarters that the *International Classification* can be used as a means of educating the medical profession into the paths of taxonomic righteousness shows a misconception of its function, and places the cart before the horse, since education through agreed nomenclature must be the first step, followed by the gradual dropping of outdated terms from the classification.

The logical approach seems to be to achieve an agreed nomenclature and use this for the main group headings, and then decide under which of these groups various alternative diagnostic terms should be placed.

Table 1 shows the classification of the «Effects of heat and insolation» given in the present edition of the *Manual of International Classification of Diseases, Injuries and Causes of Death*. Prickly heat and anhidrosis are classified under «Diseases of sweat and sebaceous glands», but there is no category for mental illness due to heat. Diagnoses such as heat neurasthenia and heat asthenic reaction have to be classified under the general headings of neurasthenia and asthenic reaction.

It will be noted that the heading of the category N981 is «Effects of heat and insolation», the latter term meaning exposure to the sun's rays. I doubt if anyone here still subscribes to the conception contained in the term, sunstroke, which leaves only sunburn as an «effect of insolation». It would seem better to categories sunburn elsewhere and to reserve N981 for «Effects of heat».

Work over the past twenty years has made it clear that the term heat exhaustion, without any qualification, is too vague for clinical use and includes a number of conditions whose epidemiology, clinical presentation, and treatment are quite distinct.

Aware of these shortcomings of the *International Classification*, and prompted by various interested parties, the Climatic Physiology

Committee of the Medical Research Council had a new classification prepared which could be used within the framework of the present *International Classification* pending more fundamental revision in 1967.

TABLE 1

N981 EFFECTS OF HEAT AND INSOLATION

This title excludes burns due to hot substances and flames (N940-N949) and diseases of sweat glands due to heat (714.0).

N981.0 *Heatstroke, except sunstroke*

Heat apoplexy	Thermoplegia
Heatstroke	—

N981.1 *Sunstroke*

Ictus solaris	Sunstroke
Insolation	Thermic fever

N981.2 *Heat cramps*

Heat cramps	—

N981.3 *Heat exhaustion and collapse*

Heat collapse	Heat prostration
Heat exhaustion	Heat syncope

N981.4 *Sunburn*

Actinic dermatitis	Sunburn

N981.5 *Other*

Excessive heat	Heat effects NOS due to: hot weather overheated places

The Medical Research Council's proposals (Table 2) were published with an appendix giving a schedule of heat disorders in the *British Medical Journal* of 28th June 1958. The schedule defined, by means of a short etiological and clinical description, each of the disorders listed in the classification. The modifications and additions were shown by letters, as required by international agreement. Sunstroke (981.1) had been omitted on the grounds that it was no longer recognised as an entity. The numbers in the second column were intended for private use only, since the numbers 981.6 to 981.9 are not officially a part of the international classification.

TABLE 2

Title	I.S.C. number[1]	Modified number (for processing but not for publication of statistics)
Heat stroke Heat hyperpyrexia — Following anhidrosis, febrile illness, or unqualified	N981.0 N981.5a	N981.0
Heat cramps	N981.2	N981.2
Heat exhaustion:		
1. Anhidrotic	N981.3a N981.3	N981.6
2. Salt-deficiency (excluding heat cramps).	N981.3b	N981.7
3. Unqualified (including water-deficiency heat exhaustion, exercise-induced heat exhaustion, and heat syncope)	N981.3c	N981.3
Sunburn	N981.4	N981.4
Prickly heat.	714.0a	N981.8
Anhidrosis	714.0b	N981.9
Heat neurotic reaction (including chronic heat neurotic reaction, chronic heat fatigue, mild asthenic reaction, mild heat fatigue).	318.3a	318.3 (part)
Other heat effects (including heat oedema)	N981.5b	N981.5

[1] In accordance with the codes of the *Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death* relating to heat illnesses (1957, W.H.O., Geneva).

In formulating this classification the Committee were hampered by the necessity to work within the framework of the existing classification and therefore had only four spare numbers at their disposal.

I propose now to examine the M.R.C. classification and consider what improvements might be made given the clean sheet available when the next revision is made.

The separation of anhidrotic and salt deficiency heat exhaustion seems to me to be well founded, and once salt deficiency heat exhaustion has been given a distinct entity, there seems little justification for retaining heat cramps. There is a body of opinion in favour of giving a separate category to heat exhaustion due to pure water deficiency, since it is becoming increasingly recognised that under certain circumstances people may develop heat exhaustion through simply not drinking enough water.

Exercise induced heat exhaustion and heat syncope seem to me to be wrongly placed under the title «Heat exhaustion». Exercise induced heat exhaustion is exactly the same as exercise induced exhaustion in temperate climates, except that under hot conditions it is likely to be induced by a smaller amount of exercise. Exhaustion due to excessive exertion already has a category of N998, and cases occurring in hot climates should, in my opinion, be categorised under this number.

Syncope bears no etiological or clinical relationship to the heat exhaustions and does not differ in the tropics from syncope in temperate cilmates, except that it is more common. There is a category for syncope (782.5) in the *International Classification* and the question which remains to be decided is whether heat syncope warrants inclusion as a separate entity in the section on the effects of heat. Heat syncope may occur as a result of posture or of relatively mild exercise, especially in the unacclimatised, and my own feeling is that it is of such common occurrence that its separation is justified.

Prickly heat and anhidrosis are at present categorised together under «Diseases of sweat and sebaceous glands» (714.0). I consider them both to be sufficiently common and of adequate importance to justify each having a separate category, and I suggest that a distinction should be made between congenital anhidrosis and the acquired form. Whether these should come under the section on «Skin and cellular tissue» as at present or be categorised under the «Effects of heat» is a matter for convenience, and since there are the figures 714.3-714.9 vacant, it might be politic to leave them in that section.

Sunburn, as already mentioned, should be recategorised in another section, while sunstroke, although recognised by the informed as being non-existent, is still diagnosed. It should therefore be relegated to the dustbin category of «Other».

TABLE 3

N981 EFFECTS OF HEAT

This title excludes burns due to hot substances and flames (N940-N949), diseases of sweat glands due to heat (714.3 and 714.4), and mental illness due to heat (326.3, 318.6 and 790.1).

N981.0 *Heatstroke and heat hyperpyrexia*

Heatstroke	Heat apoplexy
Heat hyperpyrexia	Thermoplegia

N981.1 *Heat exhaustion and collapse, anhidrotic*

Anhidrotic heat exhaustion	Anhidrotic heat collapse
Tropical anhidrotic asthenia	Thermogenic anhidrosis
Heat exhaustion type II	—

N981.2 *Heat exhaustion and collapse, salt deficient*

Salt deficient heat exhaustion	Heat cramps Heat exhaustion type I
Salt deficient heat collapse	—

N981.3 *Heat exhaustion and collapse, water deficient*

Water deficient heat exhaustion	Water deficient heat collapse

N981.4 *Heat exhaustion and collapse, unqualified*

Heat exhaustion	Heat prostration
Heat collapse	—

N981.5 *Other*

Sunstroke	Heat effects NOS due to: hot weather overheated places

N981.6 *Heat syncope*

Postural heat syncope	Exercice induced heat syncope

TABLE 4 — *Heat illness not categorised under N981, effects of heat*

318 PSYCHONEUROTIC DISORDERS, OTHER, MIXED, AND UNSPECIFIED TYPES

318.6 *Heat neurotic reaction*

Heat neurotic reaction	Heat neurasthenia

326 OTHER AND UNSPECIFIED CHARACTER, BEHAVIOUR AND INTELLIGENCE DISORDERS

326.3 *Acute situational maladjustment*

Abnormal excitability under minor stress	Combat fatigue Operational tatigue
Acute situational maladjustment	Acute heat fatigue

790 NERVOUSNESS AND DEBILITY

790.1 *Debility and undue fatigue*

Asthenia	Not of newborn, and not senile
Cachexia	
Debility	
Undue fatigue	
Chronic heat fatigue	

This title excludes combat fatigue, operational fatigue, and acute heat fatigue.

714 DISEASES OF SWEAT AND SEBACEOUS GLANDS

714.3 *Anhidrosis*

Congenital anhidrosis,	Acquired anhidrosis

714.4 *Prickly heat*

Prickly heat	Miliaria rubra
Heat rash,	—

714.5 *Other diseases of sweat glands*

Other diseases of sweat glands

We now come to the most difficult of all the heat categories — the mental effects of heat. In the present *International Classification* there is no category assigned to mental illness due to heat. The M.R.C. Classification suggests «Heat neurotic reaction». In my opinion there are three distinct mental conditions caused wholly or partially by heat and I suggest the following nomenclature:

1. *Acute heat fatigue.* — Attributable to short exposure to a very hot or ill-ventilated environment, and resulting in falling off in performance of skilled and semi-skilled work.
2. *Chronic heat fatigue.* — Attributable to long exposure to a hot environment and its accompanying social and other factors, and manifested by deterioration in performance, irritability, depression and sleeplessness. This is the condition from which almost all Europeans suffer towards the end of a tour of duty in the tropics, and in no way denotes any psycho-neurotic disposition.
3. *Heat neurotic reaction.* — A definite psycho-neurosis precipitated by working in a hot environment, from which the patient is likely to recover on transfer to more agreeable environmental conditions.

With the foregoing concepts in mind I have devised Tables 3 and 4 as a suggested basis for the classification to be adopted in 1967.

The mental illnesses have been given numbers in the appropriate sections outside the 981 series as a matter of convenience, but should of course be grouped with the other heat effects in a nomenclature.

Finally I would emphasise once again that the function of a classification is to sort diagnoses in current use and is not concerned with terms, however appropriate, which are not in fact used. If therefore members of this Congress agree with the nomenclature suggested or reach agreement on some alternative nomenclature during the course of discussion, it is essential that every effort should be made to standardise teaching through textbooks, medical schools and nomenclatures of diseases, so that the approved terms will become common usage and others will fall into disuse.

ANIDROTIC HEAT EXHAUSTION

WILLIAM LADELL

Anidrotic heat exhaustion is the least dramatic and most insidious of the disorders due to heat. It was first recognised as a distinct clinical entity during World War II, when cases were reported from New Guinea (Allen & O'Brien, 1944), the Southern States of America (Wolkin, Goodman & Kelly, 1944) and by my colleagues and myself (Ladell, Waterlow & Hudson, 1944) from Iraq. The common story in all cases was of a progressive physical exhaustion coming on after fairly prolonged sojourn in a hot climate associated with a diminution or even a complete failure to sweat. The sweating defect varied from an almost complete cessation in all parts except the face, reported by Wolkin *et al.*, to relatively minor diminution on arms and legs only, reported by us in milder cases. Our patients complained of a polyuria, but this was not reported by the Americans; Horne & Mole (1950), however, reporting from India, did find this. Both the Australian workers and ourselves noted an association between the condition and healed prickly heat, but this was not reported by the Americans. In all cases the only effective treatment was rest and removal to a cooler climate. After reconciling the various reports, the clinical picture emerges of a man who has spent weeks or months in a hot climate and apparently becomes well acclimatized, complaining of a weakness and exhaustion exacerbated by heat which has come on for no apparent reason or extra stress; he feels all right in the cool of the early morning, but as soon as the temperature gets up he collapses. There are usually complaints of dizziness, shortness of breath, and loss of appetite; he also reports that he is sweating less than he used to, but on the other hand he is producing very much more urine, and is

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having to empty his bladder nine times or more daily. He may give a history of having recently had severe «prickly heat» *(Miliaria rubra)*.

DIAGNOSIS AND TREATMENT

From examination of the cases I have seen there appeared to be little obviously wrong; there were no clinical signs of dehydration. The skin was flushed and warm to the touch; there were usually patchy dry areas, but never on the face; these areas often showed signs of healed prickly heat, with a branny desquamation and some lichenification. The systolic blood pressure was a little raised, but the diastolic low and difficult to measure. They did not faint with passive standing. The rectal temperature was usually above 38°C (100.4°F). The skin temperature in many cases was within 0.5°C of the rectal, whereas in normal controls under the same conditons the difference was 2.0°C or more. Daily urine output averaged 2,700 ml, while in hospital and the S.C. was below 1,010. There was a mean daily urinary chloride loss of 3.5 g (as $NaCl$) suggestive of good electrolyte balance, but the mean plasma chloride content was only 90 meq./l. Measurements of plasma volume and thiocyanate space indicated some slight water as well as electrolyte deficiency. Blood urea levels were normal or slightly low. When water and salt were given to these patients on their first admission to correct these deficiencies, they failed to respond and the additional salt and water was excreted in the urine. Samples of sweat collected in full length bags from patients in the wards had a higher electrolyte content than that of control healthy subjects. Only rest in a cool air conditioned ward made any difference to these men; even after two or three weeks their sweating might not be back to normal, but they would begin to retain water and salt and the chloride content of their plasma would return to normal somewhat sooner. On re-exposure to the heat outside relapses might occur and some of our patients were readmitted after apparent perfect cures several times.

It was sometimes very difficult to distinguish these cases from malingerers; the history of diminished sweating, especially if there had been prickly heat, was the most important point, but this diminution might not be very obvious in a normally heavy sweater. Even the rectal temperature increase could be misleading as under hot conditions, especially if the subject has been exercising, a slightly raised body temperature is quite normal; but in a normal subject this rise is not maintained, whereas in the anidrotic patient it is. It is on this fact and on the history of poor sweating, which may be confirmed by a low skin/rectum temperature gradient, that a diagnosis must be made; failure to diagnose and to remove from the environment to which the patient is no longer adapting (which is what «treatment» really amounts to) can be disastrous, as in some cases this condition can develop into a mild hyperpyrexia, the so called «transitional» cases described by us in Iraq. So far as we know, however, such cases never went on to frank «heat stroke». The clinical experience of the other workers was substantially the same as ours.

THE NATURE OF ANIDROTIC EXHAUSTION

But for one complicating factor the genesis of anidrotic heat exhaustion would appear to be quite simple; for some reason, usually prickly heat, sweating is diminished, evaporative cooling is defective, the body temperature rises, and the patient becomes exhausted. The complicating factor is the failure to retain water and salt even though the body is deficient in these materials; moreover the tachycardia, the raised blood pressure and the weakness, which is very real, are all more than would be expected from the relatively small rise in body temperature. In addition to the local skin changes therefore there must be general systemic ones as well.

LOCAL SKIN CHANGES

The skin changes in prickly heat have been extensively studied by Shelley, Horvath, Weidman & Pilbury (1948), and the miliarias in general have been reviewed recently by Sargent and Slutsky (1957). After prickly heat there is a definite falling off in the number and activity of the sweat glands. Other skin trauma, for example resulting from ultraviolet irradiation (Thomson, 1951*a*, *b*), will also reduce the rate of local sweating. The overall rate of sweating is also reduced; observations on a patient both before and after a severe attack showed that in healed prickly heat not only was the sweat less at a given rectal temperature than before, but also that the rise in body temperature was greater in a given heat stress, even though enough sweat was being produced to give adequate cooling (Ladell, 1951). This might be indicative of a systemic failure, but according to the concept of «wetted area» (Gagge, 1937) or skin relative humidity (Mole, 1948) for a given heat loss in a given situation there must be a certain excess of sweat; a higher rectal temperature in a given situation indicates that the sweat glands are not only less active, but also less responsive, as the stimulus must be greater to produce the same amount of sweat.

SYSTEMIC EFFECTS

The outstanding systemic changes are weakness, low plasma chloride and inability to retain salt. This combination is suggestive of adrenal cortical insufficiency, while the polyuria and failure to retain water is suggestive of posterior pituitary insufficiency and diabetes insipidus. Hare, Hickey and Hare (1941) showed that with prolonged dehydration the antidiuretic content of a rat's pituitary is reduced, and their results have subsequently been confirmed by many workers. On the hypothesis, therefore, that in anidrotic asthenia there might have been a similar pituitary fatigue due to the prolonged physiological strain of the hot summer, some patients were treated with pituitrin, but this failed either to reduce the urine flow or to increase its concentrations. Some, but not all, cases responded to treatment with the synthetic corticoid desoxycorticosterone acetate (DOCA), however, by retention of chloride. If, therefore, anidrotic asthenia was an endocrine disorder replacement therapy in our cases was not effective, but later Horne and Mole (1951) claimed that in their cases the polyuria was sometimes controlled by pituitrin.

ANIDROTIC EXHAUSTION AS A «STRESS» REACTION

Selye's «General adaptation syndrome» postulates a «stage of alarm» (Selye, 1949) in response to any non-specific stress. Following this there is an increased secretion of both the mineralo- and gluco-corticoids; with consequent retention of electrolytes and increased nitrogen metabolism, which is the stage of resistance. With long continued or extra stress the «stage of exhaustion» sets in; the capacity of the adrenal cortex to excrete comes to an end and the compensatory steroid effects are lost. Selye points out that in the «shock» and resistance phases «adaptation to one agent is acquired at the expense of resistance to others». The normally fully acclimatized man in a hot climate could be considered to be in the «stage of resistance» to the stress of the climate; the additional stress of a severe skin lesion such as prickly heat might be enough to initiate the «stage of exhaustion». There would then be a deficiency of corticoid secretion, and specifically of aldosterone; this lack of mineralo-corticoid would account for the low plasma electrolyte and for the failure of the renal tubules to reabsorb sodium chloride. It would also account for the high salt losses in the sweat as a low electrolyte content of the sweat is dependent upon the adrenal cortex (Ladell, 1945; Conn, 1944, 1949 and pers. comm.). In support of this hypothesis is the observation that out of four cases treated by us with DOCA three responded with an increased salt retention. The fourth case, however, showed little or no response, which suggests «failure of the target organ» (Ingle, 1953).

ANIDROTIC EXHAUSTION AND THE PITUITARY

In contrast to Horne and Mole's patients, none of the cases seen by us responded to antidiuretic hormone. The high rate of urine flow and the low S/G were maintained despite the administration of doses of pituitrin sufficient to give rise to abdominal discomfort. At times, therefore, there is clearly another «failure of the target organ», in this case of the renal tubules. This failure may explain several of the manifestations in anidrotic exhaustion. If the kidney no longer responds to the ADH, then a watery diuresis will follow; this itself will be conducive to electrolyte loss (Wolf, 1945). If the rest of the bodily mechanisms for the proper control of the volume and osmotic content of the body fluids continue to function, the lack of response by the kidney would result in the continued secretion of the hormone, so that there would be a high concentration of ADH in the blood, though kept partially under control by detoxication in the liver and loss in the urine. Hellman and Weiner have in fact shown (1953) that ADH is found in the urine during exposure to severe heat.

The effect of ADH on the overall sweat rate of profuse sweaters is to reduce it (Ladell, 1948) and more recently we have shown (Whitcher and Ladell, unpublished work) that after pituitrin the response of the sweat glands, expressed as the ratio (sweat rate/rectal temp. rise), is also reduced. The same reduction is seen after prickly heat. The experimental injection of pituitrin, however, makes the skin pale, whereas in the anidrotic patient the skin is flushed, but this may not be a significant difference, as other factors, as suggested below, may be concerned in the skin disorder. On this hypothesis both the polyuria and the anidrosis may be explained.

SWEAT GLAND FATIGUE

It has been suggested by some workers that the anidrosis is a chronic condition of «sweat gland fatigue». The chief argument against this is that the classic «decline of sweating after severe heat», first described by Gerking and Robinson (1946), is reversible; there have been no indications of a chronic effect. The falling off in sweat rate during an exposure to heat is associated with a high rectal temperature and when the rectal temperature falls, the sweat rate recovers again (Ladell, 1957). The «peak» rectal temperature above which the overall sweat rate falls is, however, probably 1°C higher than the temperatures usually found in anidrotic exhaustion (Ladell, 1955). There is nevertheless a progressive diminution in the sweat gland response (as defined above) as the body temperature rises above normal. There is some evidence (Ladell, 1958) that this diminution in response, which eventually manifests itself as an apparent «fatigue», is due to a diminished sensitivity of the glands to the neuroglandular transmitter, acetylcholine; such a diminution in sensitivity with a rise in temperature has been demonstrated by Thaysen and Schwartz (1955) towards intradermal acetylcholine. The same diminution in reactivity might occur irreversibly after prickly heat. The reduced response after prickly heat to an overall heat stress does point to a reduced responsiveness of the sweat glands to the central stimulus, but whether this is another example of «failure of the target organ response» cannot yet be substantiated. Thomson (1951*b*) reported a reduced response to iontophoresed acetylcholine in skin after ultraviolet irradiation; but this; as he points out, could have been due to physical blockage of the sweat ducts.

The above considerations apart, the falling off in sweat gland response with increasing rectal temperature is the reason why anidrotic exhaustion is dangerous. It may be assumed that the pattern of the response will be the same even in disease, hence even if the defect is of capacity, and not of reactivity, there will be falling off in the total sweat output once a certain «peak» rectal temperature has been reached. If the sweating capacity is already reduced almost to the minimum required to maintain thermal equilibrium, any extra metabolic effort or thermal stress will outstrip the cooling powers of the body, the rectal temperature will rise further, and the peak may be passed; once this occurs, any further temperature rise will result in even less sweating and so hyperpyrexia may develop.

ANIDROTIC EXHAUSTION A COMPLEX DISORDER

Anidrotic exhaustion cannot be explained on the basis of only one disorder. Each of the factors mentioned above explain some, but not all, of the changes seen. Prickly heat may cause a reduced sensitivity of the sweat glands to acetylcholine and it may also reduce the capacity of the fully reactive gland to secrete, but these changes do not explain the systemic manifestations of anidrotic exhaustion. Failure of the adrenal cortex in the «stage of exhaustion» would account for the electrolyte deficiencies, the high electrolyte content of the sweat. and the asthenia, but it would not explain the polyuria or the anidrosis. Failure of the renal tubules to respond to ADH would explain the polyuria, and any resultant hypersecretion by the pituitary might account for the anidrosis; the electrolyte losses might be attributed to the polyuria, but this still leaves the asthenia unexplained. The explanation probably lies with all three disorders: the sequence of events may be (Ladell, 1957):

1) Continued secretion of ADH, in response to heat, and of aldosterone secretion, in response to high electrolyte demands.
2) Failure of the renal tubules to respond to ADH, leading to excessive water losses with more ADH production.
3) ADH in excess partially inhibiting sweat production.
4) Further inhibition of sweating by prickly heat, resulting in inadequate control of body temperature.
5) The inadequate body cooling giving additional stress so that the adrenal «stage of adaptation» gives way to the «stage of breakdown», leading to exhaustion with general diminution in adrenal secretion.

In this scheme prickly heat is not the primary event; it is shown as the extra stress, which finally precipitates the breakdown. Prickly heat could itself be an early indication of adrenal insufficiency, in which case it becomes a symptom and not an aetiological factor. It is possible that in adrenal insufficiency the skin becomes more sensitive to the sweat-saturation which leads to prickly heat (Shelley *et al.*, 1948); the therapeutic and prophylactic importance of the adrenal corticoids in skin disorders is well known. The effect of salt intake on prickly heat, reported by Horne and Mole (1949), might be explained along similar lines; with excess salt intake there is depression of adrenal cortical secretion, and this would increase the susceptibility of the skin to local disorders.

There are some complicating factors that make it difficult to accept this simple hypothesis except as a basis on which further work may be planned; in particular there are the good responses to pituitrin reported by Horne and Mole (1949) and our own failure, in one instance, to get a response to DOCA. Anidrotic exhaustion is not, however, a disorder that can be reproduced experimentally and detailed investigations must be deferred until actual cases are available for study. But from the foregoing considerations it would appear that the outstanding questions to be answered are:

1. What are the responses of the patient with anidrotic exhaustion to:

    a) Aldosterone therapy?
    b) Pituitrin therapy?

    Do all patients respond in the same way?

2. What are the levels in the urine (and ideally in the blood) of.

    a) Aldosterone?
    b) Antidiuretic hormone?

3. Does the response of the sweat glands to intradermal acetylcholine alter after prickly heat?

More work is also required on the effect of pituitrin on the sweat rate. The possibility cannot in any case be excluded that there may be several minor variations of the anidrotic exhaustion syndrome and this might be the reason for the anomalous results of treatment and the slight differences in the clinical pictures reported by various workers.

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AETIOLOGY OF HEAT ILLNESS IN KUWAIT

J. GUTHRIE, M.B., CH.B.

I intend only to offer some impressions and opinions arising from 10 years general medical practice with Kuwait Oil Company in Kuwait. These have been influenced by some simple investigations we have carried out in the last 3 or 4 years. We began these by enquiring into shipboard conditions, and the habits of ships crews aboard oil tankers calling at Kuwait during the summer months. In 1955 and 1956, questionnaires were completed for the more severe cases of heat illness amongst these men. Replies were sometimes unreliable, the numbers were small (40 and 45), and, because of limited facilities at the Port First Aid Post, diagnoses were mostly clinical and unsupported by further studies. Nevertheless, some interesting information emerged, some of which is presented here. Shortly after this small beginning, a full and independent study of environmental conditions in Kuwait Oil Company areas was undertaken by the Liverpool School of Tropical Medicine under the directorship of Professor B. G. Maegraith. Since the summer of 1956, a research team headed by Dr. Charles Leithead has come out to Kuwait each year, from June to September, to carry out detailed studies. This work is continuing.

The State of Kuwait lies at the head of the Persian Gulf. The summer climate in the hinterland is extremely hot and dry, though occasional humid spells occur. Shade temperatures range between 110°F in June and 122°F in August, with relative humidity varying between 25 % and 65 %. On the coast, temperatures are about 5 degrees lower, and the humidity between 30 % and 90 %. A northerly prevailing wind somewhat mitigates the heat between June and August.

The types of heat illness most commonly encountered are heat exhaustion of salt and water depletion type, and heat hyperpyrexia.

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Anhidrotic heat exhaustion is sometimes seen amongst ships crews. Heat syncope occurs occasionally during humid spells in mid-summer.

In Kuwait Oil Company operations we have two communities. One lives and works seven miles inland in the Oil Company town of Ahmadi, or on the coast at the oil port Mena-al-Ahmadi. The second community comprises the crews of visiting oil tankers. Since conditions differ considerably between these two groups, they will be taken separately. The ships crews will be considered first.

Approximately 250 tankers of many nationalities call each month at Mena, which is the world's largest oil terminal. Over the year the total number of sailors in these ships exceeds 170,000.

By far the greatest numbers of heat illness cases seen in Kuwait occur amongst these tanker crews. In the last ten years, at least 18 cases of heat hyperpyrexia have been taken off tankers at Mena-al-Ahmadi. Each summer since 1951, from 60 to 200 cases of heat illness have been diagnosed amongst sailors reporting at the Port First Aid Post.

The voyage from Suez to Kuwait between June and September is one of the most trying in the world. Two thirds of it is made in the intense humid heat of landlocked seas, the Red Sea and the Persian Gulf. During the middle third, in the open Arabian Sea, the high temperatures are somewhat reduced by the South West monsoon.

From the moment he enters the Red Sea, where the temperature and humidity rise sharply within twenty-four hours, until his ship ties up about ten days later at Mena-al-Ahmadi Pier, the sailor probably never ceases to sweat. Intense radiant heat from the sun, decks, superstructures, and ship's engines, and a stifling atmospheric humidity, form his inescapable environment, unless his ship is one of the few aircooled tankers. A heterogenous collection of sailors is exposed to these climatic conditions, some unacclimatised apprentices, others acclimatised veterans, some young, others middle aged, some healthy and physically fit, others unfit, and a few suffering active illness.

To the primary aetiological factor of heat stress are added others arising from shipboard conditions and from the habits of the crews themselves.

Despite much improved crews quarters in the newer ships, the great majority of tankers still totally lack airconditioning, and heat stress therefore continues to be severe. Cooling, even of mess decks and recreation rooms, would considerably relieve the heat load, and benefit especially those working in engine rooms and galleys, where heat ilness occurs two or three times more frequently than above decks.

In our two questionnaire series more than 70 % stated that they drank 8 pints or less of water daily. For men sweating during most of the twenty-four hours, even double this quantity would be insufficient.

In one questionnaire series 55 %, and in the other 71 %, stated that during the voyage they had taken 3 g or less of extra salt daily. None took more than 8 g. This is a low intake for extreme conditions even in the light of recent work, by Conn and others, though Ladell (1957) has challenged the traditional view that large salt replacement is necessary.

Alcohol habits were enquired into. Unfortunately most replies were evasive or unreliable, though a few admitted to 1 l of wine daily. In several cases of heat hyperpyrexia we have had reason to believe that alcohol in excess was a precipitating factor. In one fatal case for example, the patient had taken 3 l of red wine with his mid-day meal a few hours previously.

In the belief that much heat illness on tankers is due to ignorance of the hazards involved, we began three summers ago to issue a pamphlet to the crews of all visiting tankers. This was translated into 8 languages, and gave simple information about heat disorders and their prevention. 20,000 pamphlets were distributed during the first season and we are continuing to issue them each summer. It is possible that they have had some effect. Crews seem to show a genuine interest in them, and the cases of heat illness diagnosed at the Port First Aid Post for the last three years have numbered 222, 137, and 63, though the number of visiting tankers has not decreased.

One other factor has greatly interested us. Heat illness amongst tanker crews appears to be more common during the voyage up the Gulf in ballast and against a head wind than during the return trip under less favourable conditions, with tanks full of oil at a temperature of perhaps 120°F and the prevailing wind now a following one, with therefore little air movement. Possible explanations of this apparent anomaly are that sailors on the upward voyage are inadequately acclimatised. At Mena-al-Ahmadi the more severe cases will be removed from their ships and taken to hospital. Those returning, having acclimatised successfully, are fit to endure the downward voyage.

Conditions ashore are in sharp contrast to those afloat. The shore based community numbers some 25,000. Of these about 9,500 are American, Arab, British, Indian and Pakistani employees of Kuwait Oil Company. The remainder are their dependants. To these must be added about 7,000 labourers, mostly expatriate Arabs, working for contractors and living in nearby villages.

About 15 % of employees live in aircooled houses. Many of these work for 8 hours or more each day in the heat of the sun. Of the remaining 85 % of employees, many work in aircooled offices, but live in uncooled houses. No case of heat hyperpyrexia has ever occurred amongst these employees, and heat exhaustion is rare. There is no doubt that aircooled living or working conditions, together with sustained medical propaganda, provide sure protection against heat illness.

Among Company employees living in uncooled houses and working out of doors or in hot indoor conditions, heat exhaustion in previous years was seldom seen, and heat stroke has claimed only 2 victims over the last ten years. One of these was an Indian cook working in a hot kitchen, the other was a Kuwait Arab who fell asleep in the mid-day July sun.

During the last two or three years, as many as 10,000 Arabs, Iranis, Iraqis, Lebanese, Palestinians and Syrians working for contractors, have been employed at some periods upon large scale construction projects. In the summer of 1957, 157 cases of heat exhaustion were diagnosed amongst this group. 141 of these were expatriate Arabs, most of whom were working on the construction of a refinery, where radiation from large metallic structures was considerable.

A point of great interest to us is that we are now beginning to see heat exhaustion amongst Kuwait men. In the summer of 1957, 16 such cases were diagnosed. Hitherto the Kuwaiti Arabi, wonderfully adjusted to his climate both physiologically and by mode of life, has seemed to be immune to the heat, but the advent of industrialism, with plenty of work at good wages, is changing the old way of life. Nowadays the Kuwaiti, instead of resting during the heat of the day, is doing heavy work. There is a strong clinical impression that he is not adjusting his water intake to the new conditions, and that this is leading in some cases to water depletion. Urinary salt excretion is high amongst Kuwaitis as a group, but salt depletion occasionally occurs, perhaps because many Kuwaitis now drink distilled water instead of the brackish they have been accustomed to, which contains a high content of chloride.

In summary, heat illness as seen in Kuwait Oil Company operations occurs mainly amongst ships crews and expatriate Arab labourers.

The aetiological factors of importance are the severe heat stresses of the voyage from Suez to Kuwait in uncooled ships, and the unsuitable habits of the crews, many of whom are unacclimatised and ignorant of their physiological needs.

Those living or working in aircooled conditions ashore do not suffer from heat illness, and it is believed that aircooling in ships could virtually eliminate heat illness amongst ships crews.

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THE EFFECT OF THE PHYSICAL EFFORT IN THE DEAD SEA AREA

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S. MASSRY [1]
and
J. B. ROSENFELD [1]

This study is concerned with the influence of varied amounts of water intake on equal effort of walking for 2 hours at the speed of 6 km per hour, outdoor temperature being 42°C. The experiment was conducted in Sodom, in the Dead Sea Region, from 10-12 A.M., July, 1957. The place is located 395 m below sea level. The participants in the experiment were 8 healthy subjects, 18-26 years old, selected at random, all, except one, native to the Middle East, who had been residing at Sodom from 6-24 months.

We examined body temperature rectally, blood and saliva specimens were taken for chemical examination, blood pressure was measured, ECG and pulmonary function tests were performed. Urine was collected during the experiment. Due to the short period of time permitted for this lecture, the results of only 5 subjects will be presented.

The 5 subjects were divided into 2 groups:

Group A: 3 with no water intake.
Group B: 2 who were forced to drink every 20 minutes, regardless of their thirst urge.

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[2] Biochemical Department, Hebrew University, Jerusalem, Israel.

The relation of height to weight of the participants showed that all except one were somewhat underweight. The systolic and diastolic blood pressures were normal in all. After the experiment, the blood pressure remained normal. Those of Group A were irritated, complained the most of dryness in the mouth and fatigue. In contrast, those in Group B, although fatigued, were free of all other symptoms.

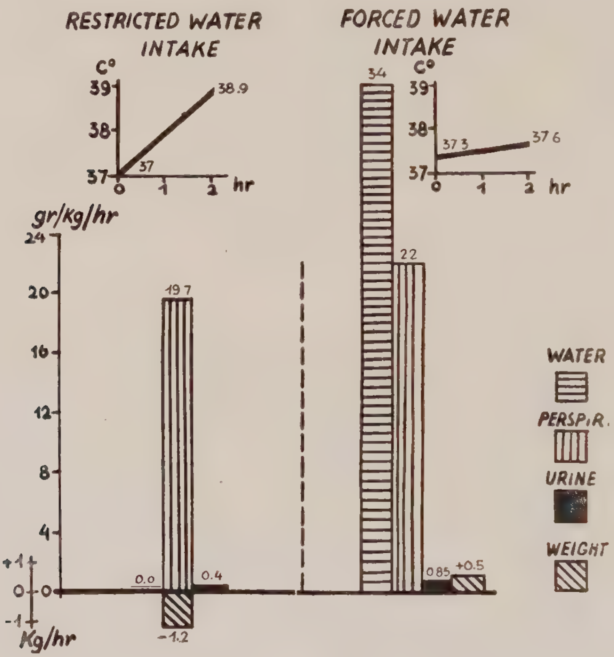

Fig. 1 — Water metabolism

The perspiration in Group A was 19.7 g/kg/hr. (Fig. 1 *). They voided 0.4 cm^3/min. They lost on the average 1,200 g/hr.

The water intake in Group B was 34 g/kg/hr. Perspiration was 22 g/kg/hr. They voided 0.85 cm^3/min. They gained 500 g in weight.

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* The results given in all the figures are expressed as averages of each group.

The body temperature in Group A increased by 1.90°C, whereas in Group B temperature practically did not change.

The average sodium values of all subjects who participated in the experiment showed hypernatremia — over 150 m Eq/L — even before the walk (Fig. 2). After the hike, the highest hypernatremia was found in Group A, whereas Group B showed a decrease, on the average of 10 m Eq/L.

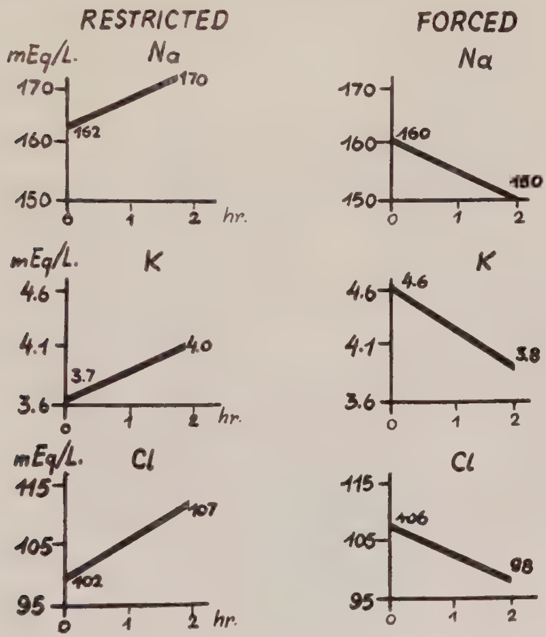

Fig. 2 — Changes in blood electrolytes following different water intake

Blood chloride changes paralleled those of sodium, but were not so conspicuous. We did not, at that time, examine chlorides of whole blood and, therefore, we cannot determine the amount of chloride shifted into the red cells. Nor did we examine the rate of chloride excretion in sweat. Potassium values of Group A at the beginning of the experiment were at the lower limit of normal and rose somewhat after the walk. In Group B, the potassium values decreased after the hike.

Urea clearance showed the same trend as creatinine clearance (Fig. 3). It was the lowest in Group A and blood urea rose accordingly. In Group B, urea clearance was higher and blood urea decreased despite the effects of the hike.

Sodium excretion in urine was lowest in the water restricted group, although blood sodium in this group was highest. Urinary sodium excretion increased in the group with fluid intake paralleling the decrease in blood sodium.

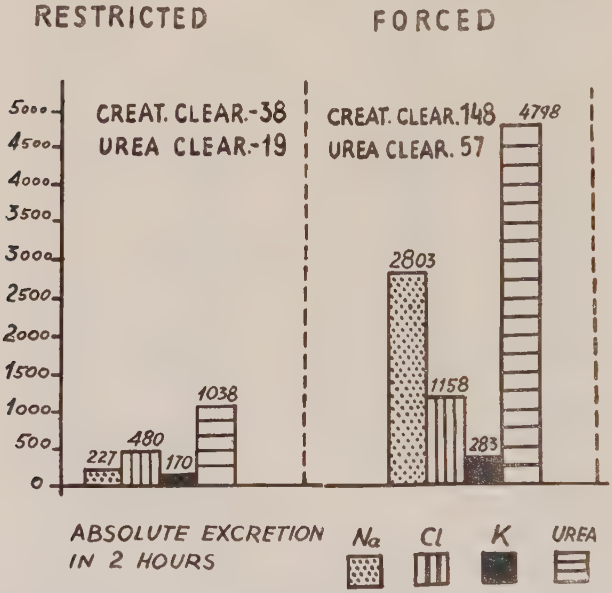

Fig. 3 — Kidney function following different water intake

The amount of chloride excreted in the urine was small in the restricted group and was higher in Group B. However, in Group A, the amount of chloride excreted was twice the amount of sodium. In contrast to this, chloride excretion in Group B was only half the amount of the sodium excreted.

As the biochemical blood values of the subjects among each group were not equal before the experiment, the end results of the average values of each group do not clearly show how each individual preserves homeostasis. We are presenting, therefore, separately the data of 2 subjects whose biochemical blood values at the beginning of the experiment were different. One of them was in Group A and one was in Group B.

Sh. M. (Fig. 4) did not drink; blood sodium before walking was in the upper limit of normal: potassium was in the lower limit of normal; bicarbonates, chloride and blood protein were within normal range; pH was 7.43.

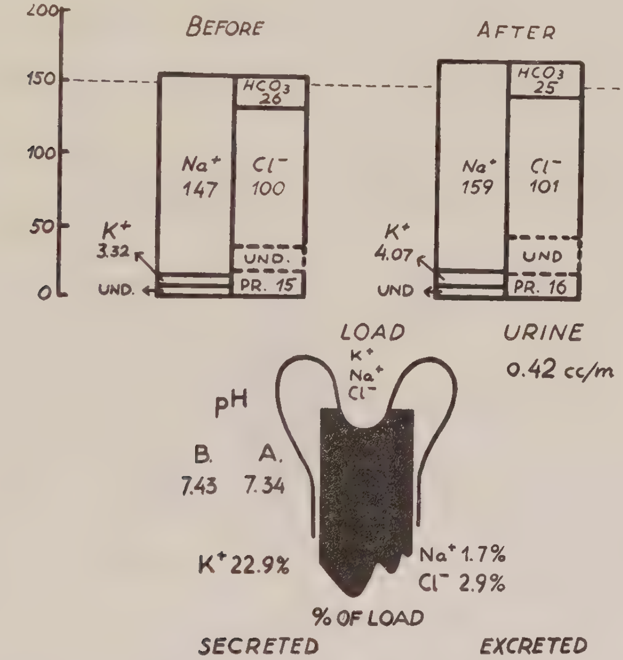
200
150
100
50
0
BEFORE
AFTER
HCO_3 26
Na^+ 147
Cl^- 100
K^+ 3.32
UND.
UND.
PR. 15
HCO_3 25
Na^+ 159
Cl^- 101
K^+ 4.07
UND
UND
PR. 16
LOAD
K^+
Na^+
Cl^-
URINE
0.42 cc/m
pH
B. A.
7.43 7.34
K^+ 22.9%
Na^+ 1.7%
Cl^- 2.9%
% OF LOAD
SECRETED
EXCRETED

Fig. 4 — Restricted water intake

After the walk, his body temperature rose. His blood sodium showed hypernatremia. The anions which had been determined did not change. During the hike he urinated 0.42 cm^3 per minute; he excreted in the urine only 1.7 % of the amount of sodium as against 2.9 % of the chloride reaching the tubulus.

Z. A. (Fig. 5) started the experiment with hypernatremia of 163 m Eq/L. Bicarbonates were 21 m Eq/L, chloride somewhat higher than the normal, blood proteins normal. In spite of walking in the heat, but drinking copiously, the hypernatremia decreased to normal (146 m Eq/L). The blood bicarbonates decreased; chloride decreased by 10 m Eq/L; blood proteins remained normal.

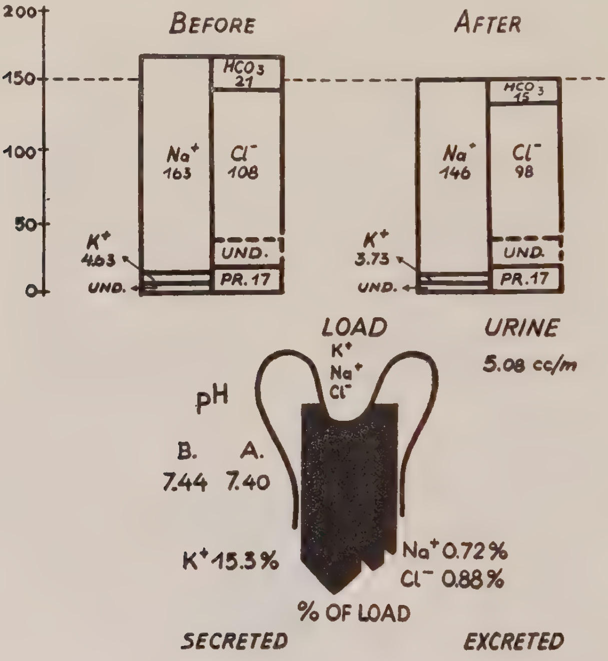
200
150
100
50
0
BEFORE
AFTER
HCO3 21
Na+ 163
Cl- 108
K+ 4.63
UND.
UND.
PR. 17
HCO3 15
Na+ 146
Cl- 98
K+ 3.73
UND.
UND.
PR. 17
LOAD
K+
Na+
Cl-
URINE
5.08 cc/m
pH
B. A.
7.44 7.40
K+ 15.3%
Na+ 0.72%
Cl- 0.88%
% OF LOAD
SECRETED
EXCRETED

Fig. 5 — Forced water intake

His urine excretion was 5 cm^3 per minute. Comparison of anion and cation excretion demonstrates a significant difference in kidney function of this subject as compared to the previous subject. Sodium excreted in urine was only 0.72 % of the tubular load; chloride excretion was almost equivalent to sodium excretion. Blood pH remained in the upper limit of the normal.

DISCUSSION

In the Dead Sea area, which is outstanding for its hot weather, one can already see at the onset of summer that the inhabitants lose weight. The water intake while working varies between 6 l to 10 l daily. They are very sensitive and are easily irritated. In the afternoon, they have difficulty in concentrating at work.

Our experiment showed that even in people native to hot areas, a 2 hour hike, the outdoor temperature of 42°C, water intake only according to thirst urge was enough to produce hypernatremia.

At this temperature, appropriate hydration, in spite of the same effort, not only can prevent hypertonic dehydration, but can also correct it. This fact proves that the hypernatremia is caused only by a greater water loss than a salt loss.

As the blood values of the participants were not equal before the experiment, it is not worthwhile to go into every result which was received from the biochemical tests. Of importance is the direction of the biochemical changes which took place in every group and the ways of preventing extreme pH changes during the development and correction of hypernatremia.

Kerpel-Fronius proved some time ago in rabbits and Nadal in humans that in water deprivation we are dealing with a diffuse body dehydration, and so the blood volume at the beginning of the process is not impaired. We also did not see striking changes in HKT; blood pressure was and stayed normal in all the subjects.

In our study, the blood volume, derived according to the formula $Vb_2 = Vb_1 \times \frac{(Hb)_1}{(Hb)_2}$, stayed normal in most of the subjects. Only in one subject with restricted water intake, along with severe hypernatremia, did the blood volume decrease by 1 l. These data support the assumption that, when losing water, the body sacrifices first of all its osmolarity in order to preserve its blood volume. Only under conditions of severe hypertonic dehydration does the body lower its blood volume.

It has long been known that dehydration causes a slight rise in the blood urea level following tissue destruction. We saw this particularly in Group A. The urea clearance in this group was also the lowest and the amount of urea in the urine was the least. In contrast to this, the group which was forced to drink excreted almost 5 times the amount of urea of the former group. This is due to the high urinary flow.

The rate of excretion of electrolytes in the urine reveals that chloride excretion in the group which did not drink is twice that of sodium, whereas in the group which was forced to drink it is less than half that of sodium. With increased dehydration and hypernatremia, urinary sodium excretion lessens whereas potassium excretion becomes relatively higher. These data resemble secondary hyperaldosteronism which arises after contraction of some critical fluid volume due to hypertonic dehydration. In Group B there was expansion of the extracellular fluid due to copious water intake. Urinary sodium excretion increased to 12 times that of Group A, whereas tubular potassium secretion decreased and was only 1/10 of the sodium excretion.

SUMMARY

In contrast to the slow development of hypernatremia following water dehydration in temperate areas, in hot climates even natives will quickly develop hypernatremia after a two hour hike. Drinking only according to thirst urge will not prevent it.

The hypernatremia is accompanied by signs which resemble secondary hyperaldosteronism. Despite the hypertonic dehydration in Group A, blood pressure and pH remained within normal limits.

Excessive forced drinking can correct and prevent hypernatremia despite the effort.

PREVENTION OF HEAT CASUALTIES[1]

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The program for preventing heat casualties in U.S. Marine Corps recruits in summer training at Parris Island, South Carolina, centers around the regulation which curtails strenuous drills during spells of hot weather, thereby reducing the recruit's metabolic heat production to maintain his total heat load within tolerable limits. Environmental heat stress is expressed in terms of the Wet Bulb-Globe Temperature (WBGT) index of Yaglou, which is derived from hourly readings of natural wet bulb, black globe, and shade dry bulb temperatures weighted and added together as follows: $0.7\ WB + 0.2\ GT + 0.1\ DB$. Significant correlation was previously demonstrated between this index and evaporative weight loss in military trainees undertaking various exercises. At index levels of 85°F or above strenuous training activities are suspended for recruits in their first three weeks of training. More seasoned recruits continue regular drills until the index reaches 88°F or above.

Other preventive measures include a policy of liberal water and salt intake, rational clothing practices, indoctrination of recruits and drill instructors in hot weather hygiene, and special conditioning platoons for obese recruits and others substandard in physical fitness.

Since its inception at Parris Island in 1953 the preventive program has undergone progressive modifications with a major change in 1956, when the WBGT index of heat stress replaced the former index, which was derived from readings of dry and wet bulb temperatures only. In the three-year period before preventive measures were introduced, the average weekly incidence rate of heat casualties during summer months exceeded 50 per 10,000. By comparison the corresponding rate was 12.4 in 1955, with a further significant drop to 4.67 in 1956, thus coinciding with the introduction of the present program. The improvement in 1956 occurred despite higher seasonal heat and at less cost in training hours than in 1955.

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[1] See also D. Minard, «Prevention of heat casualties in Marine Corps recruits. Period of 1955-60, with comparative incidence rates and climatic heat stresses in other training categories», *Military Medicine*, **126**: 261-272, 1961.

INFLUENCE DE LA CHALEUR SUR LE COMPORTEMENT RÉACTIONNEL DES VAISSEAUX PÉRIPHÉRIQUES[1]

R. LEMAIRE

Afin d'étudier les mécanismes qui contrôlent les processus de vasodilatation périphérique provoqués par la chaleur, nous avons observé les effets de cet agent sur les réactions circulatoires déclenchées, soit par la mise en jeu de réflexes vaso-moteurs, soit par l'administration de substances douées d'activité vaso-motrice connue. Nous avons exploré le comportement réactionnel des vaisseaux périphériques chez des animaux anesthésiés soumis aux effets de la chaleur.

Nous nous sommes adressés à des chiens chloralosés, placés dans une boite de chaleur. On a relevé la température de l'enceinte ainsi que plusieurs températures cutanées et la température centrale des animaux. Il a été admis que l'action de la chaleur était complète lorsque la température rectale des animaux atteignait 41°C. Pour juger du comportement réactionnel des vaisseaux, la pression artérielle fémorale a été enregistrée à l'aide d'un électro-manomètre, et le débit sanguin artériel grâce à un débit-mètre à inductance utilisé par M. B. Visscher. La connaissance des variations de ces deux grandeurs permet de calculer à l'aide de formules dérivées de celle de Poiseuille les modifications du diamètre des artères et la résistance périphérique, en admettant que dans le temps court de nos explorations le coefficient η de viscosité ne varie pas. Les investigations s'adressent aux vaisseaux de la patte postérieure du chien dont les variations de volume ont été de plus suivies par pléthysmographie. En outre, un phénomène circulatoire central a été enregistré, à titre de référence,

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[1] Travail du Laboratoire de Physiologie de l'École de médecine de Dakar.

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 105-111, 1959

sur la quatrième plume de l'appareil de Sandborn: la pression intraventriculaire du cœur droit.

Nous avons provoqué une vaso-dilatation soit par stimulation de l'extrémité centrale du nerf de Héring, soit par administration d'acétylcholine. Les vaso-constrictions ont été déclenchées par la diminution de la pression sanguine au niveau du sinus carotidien consécutive aux pincements des deux artères carotides, soit par administration d'adrénaline. Les manipulations ont été pratiquées avant et pendant l'action de la chaleur.

À la suite de la stimulation du nerf de Héring, on constate une baisse de la pression artérielle fémorale, une baisse légère du débit artériel, une augmentation, puis une diminution de volume de la patte. L'animal étant placé sous action de la chaleur, la même stimulation provoque une baisse du débit artériel accentuée, une chute de la pression encore notable, tandis que le volume de la patte ne varie plus (figs. 1 et 2). Le calcul montre que la variation de calibre des vaisseaux périphériques déclenchée par l'excitation des nerfs de Héring ne s'observe plus chez l'animal soumis aux effets de la chaleur. La résistance périphérique, qui avait diminué, suit la même évolution.

L'administration d'acétylcholine est suivie d'une baisse de la pression artérielle fémorale, d'une chute du débit artériel, et d'une augmentation très passagère, puis d'une diminution du volume de la patte. Sous l'effet de la chaleur, la diminution du débit artériel devient moins nette, tandis que l'hypotension reste marquée et que le volume de la patte ne varie presque plus. Le diamètre des vaisseaux ne se modifie plus, alors qu'il augmente avant l'action de la chaleur. Il en est de même de la résistance périphérique, qui avait diminué lors de la manipulation témoin.

Après mise en œuvre de la chaleur et aussi bien dans le cas de l'excitation du nerf de Héring que de l'injection d'acétylcholine, on observe une nette augmentation de la différentielle.

Le pincement des carotides détermine une hypertension, accompagnée d'une élévation discrète du débit artériel, tandis que le volume de la patte diminue légèrement. Le diamètre des vaisseaux diminue et la résistance périphérique augmente. L'action de la chaleur se traduit par une disparition de l'hypertension, par une franche augmentation du débit artériel et par une augmentation discrète du volume

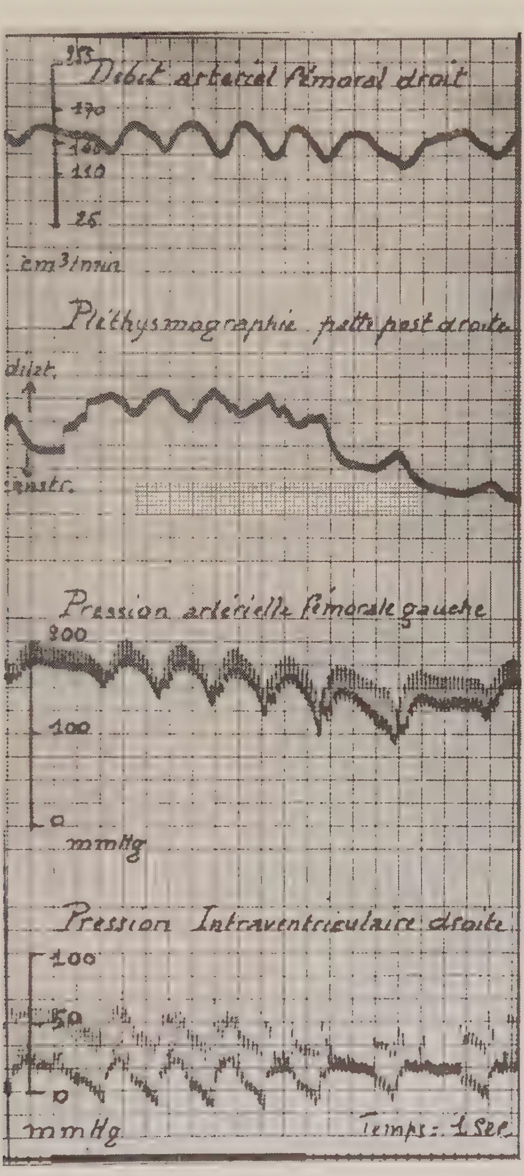

Fig. 1 — Au signal: stimulation faradique du nerf de Héring. Température centrale de l'animal: 37°C

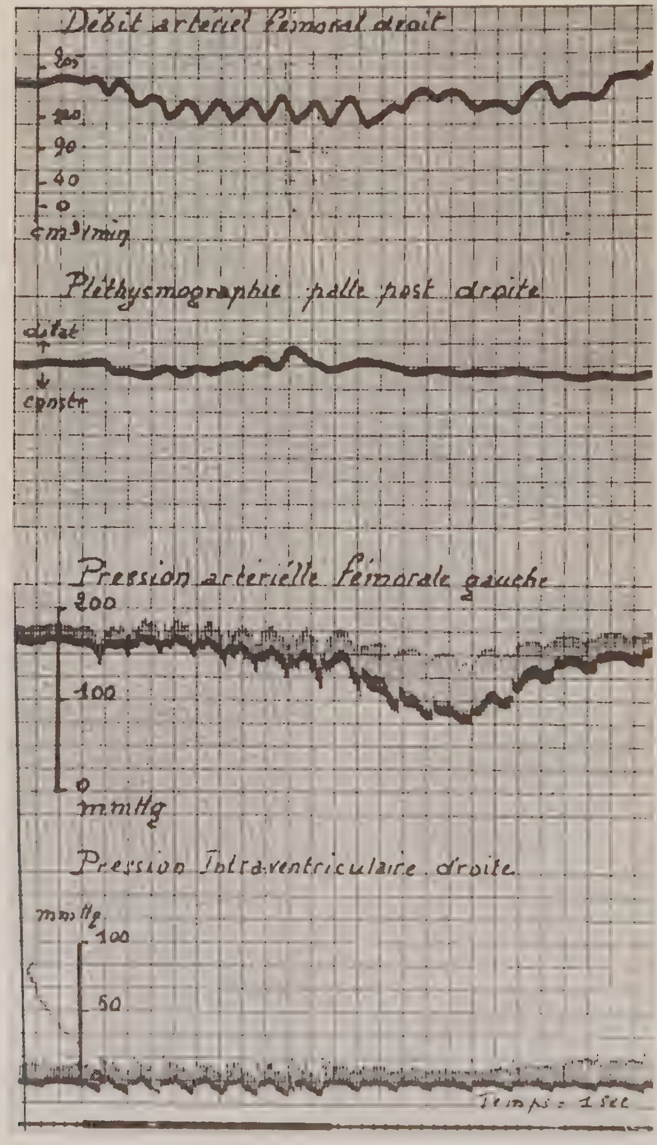

Fig. 2 — Au signal : même stimulation faradique que fig. 1. Température centrale de l'animal : 41°C

de la patte. Le diamètre des vaisseaux augmente, tandis que son inverse, la résistance périphérique, diminue.

L'administration d'adrénaline provoque une augmentation du débit artériel, une augmentation, puis une diminution du volume de la patte, accompagnant l'hypertension artérielle. Il y a augmentation de la résistance périphérique et diminution du diamètre vasculaire. Sous l'influence de la chaleur, si l'hypertension est moins nette, l'élévation du débit artériel est plus marquée, tandis que l'augmentation puis la diminution du volume de la patte sont plus discrètes. Les variations du diamètre vasculaire et de la résistance périphérique persistent, quoique moins accentuées (figs. 3 et 4).

Ces constatations soulignent tout d'abord la complexité des réactions de régulations circulatoire, les réponses étant souvent de sens contraire. La chaleur modifie ces réactions régulatrices: au cours de vaso-dilatations provoquées la capacité réactionnelle des vaisseaux périphériques paraît nettement diminuée. Au cours des vaso-constrictions provoquées, on note soit une diminution des réactions vaso-motrices, soit même une inversion. La chaleur provoque donc une dilatation des vaisseaux périphériques, qui, une fois installée, garde un caractère de permanence. Il ne semble plus possible de l'augmenter et difficile de la diminuer.

Il s'établirait ainsi sous l'effet de la chaleur une parésie vasculaire périphérique.

Cette parésie pourrait, dans une certaine mesure, rendre compte de la constatation faite par différents auteurs, à savoir: que l'on n'observe pas, chez le sujet vivant en pays tropical et pourtant soumis à de nombreuses aggressions thermiques, une hypotension notable.

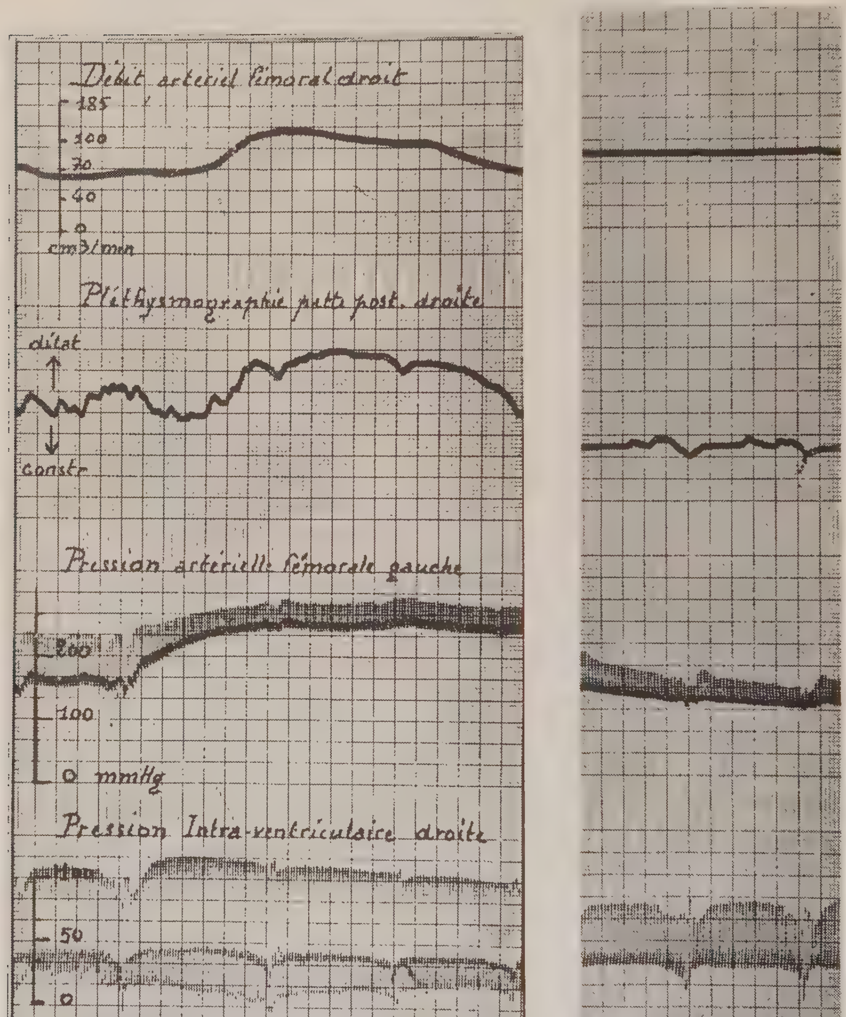

Fig. 3 — Au signal: injection intraveineuse de $^1/_{400}$ mg/kg d'adrenaline.
Température centrale de l'animal: 37°C

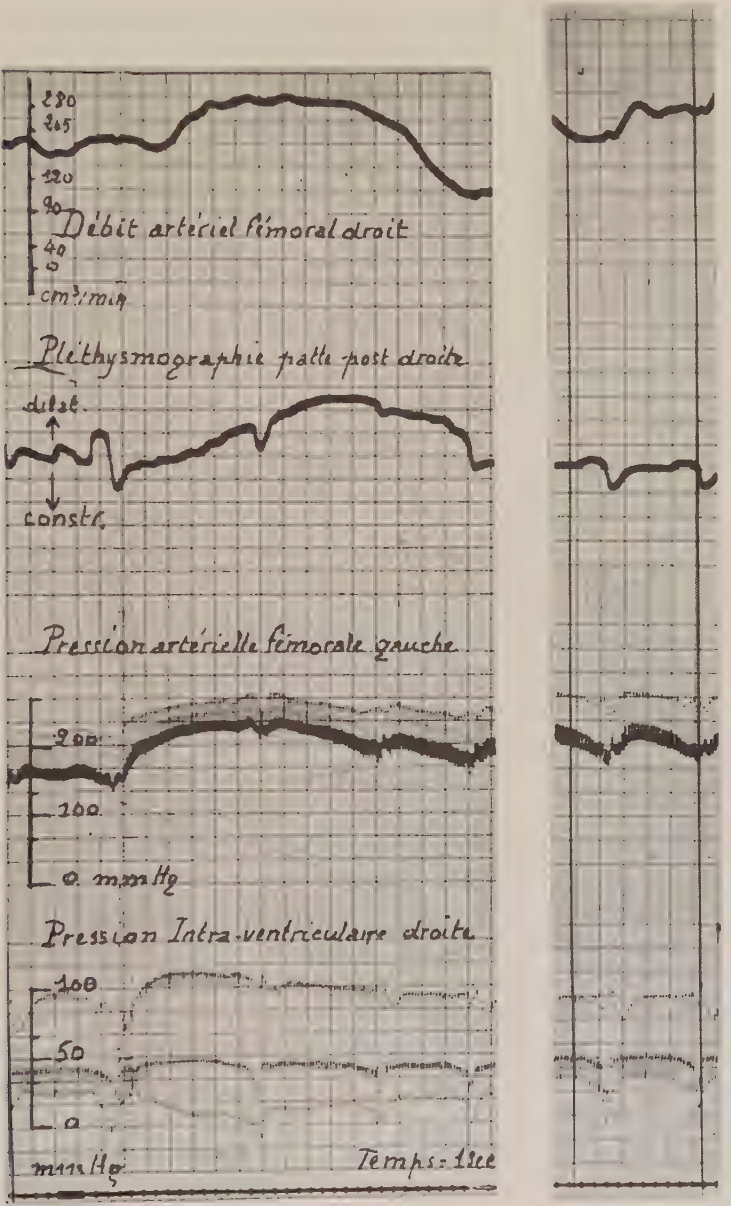

Fig. 4 — Au signal: injection intraveineuse d'une même dose d'adrenaline que fig. 3. Température centrale de l'animal: 41°C

UMA NOVA DOENÇA DE ADAPTAÇÃO

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As hemorragias gengivais dos trabalhadores indígenas são um fenómeno de há muito conhecido em Angola.

Só excepcionalmente tais gengivorragias são espontâneas. Na grande maioria dos casos deve-se, para as pesquisar, utilizar a seguinte manobra:

Toma-se o lábio inferior entre o polegar e o indicador e, exercendo uma leve pressão sobre a gengiva inferior, executam-se alguns movimentos de lateralidade; repuxando-se o lábio para baixo, descobre-se a gengiva e aguarda-se, observando, 10 a 15 segundos. Nuns casos a hemorragia surge imediata e abundantemente; noutros a sua aparição é tardia e limita-se a algumas eflorescências punctiformes de sangue junto ao sulco gengivodentário.

O que descrevemos aprecia-se bem no filme que agora se projecta.

Nas formaturas de serviçais nativos temos observado este fenómeno com bastante frequência, em percentagem variando entre 13 e 40 %.

Tais gengivorragias eram interpretadas como sintoma de carência de vitamina C, e nós próprios, nos primeiros contactos com o fenómeno, as interpretámos desta maneira.

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 112-129, 1959

No entanto, em breve fomos observando uma série de factos que nos suscitavam grandes dúvidas quanto à verdade deste conceito.

Em primeiro lugar, as hemorragias apareciam em gengivas completamente sãs e de aspecto normal. Nem o edemaciado, nem o amolecimento, nem o aspecto esponjoso das gengivas do escorbuto.

Em segundo lugar, não eram acompanhadas de qualquer outro sintoma próprio da hipovitaminose C, em especial da hiperqueratose folicular hemorrágica das nádegas e das coxas, que é uma manifestação clínica bastante precoce e frequente da carência de vitamina C.

Em terceiro lugar, se as verificávamos, de facto, em algumas regiões onde a alimentação era pobre na vitamina, também as verificávamos noutras onde os géneros alimentícios ricos nesta vitamina abundavam.

Em quarto lugar, e finalmente, verificámos que, embora as hemorragias não aparecessem, ou só aparecessem em diminuta percentagem (3 %-5 %) nos indígenas que, oriundos dum meio rural, eram inspeccionados para o trabalho, se manifestavam logo nos primeiros momentos após a sua chegada a esses locais, em percentagem muito mais elevada (13 %-40 %). Este facto estava em contradição com o que a experimentação humana sobre o escorbuto nos ensinava. Os trabalhos de Crandon (1), Farmer (2) e Mensching (3) evidenciaram, com efeito, que era necessária uma dieta absolutamente carente de ácido ascórbico, e prolongada de 4 a 5 meses, para que o escorbuto se manifestasse.

Se a consideração destes factos nos levava a admitir que as gengivorragias não resultavam de uma carência de vitamina C, dois problemas se punham então.

O primeiro consistia em provar que assim era realmente.

O segundo, em descobrir qual seria a verdadeira causa das gengivorragias.

Encarando a resolução dos dois problemas simultâneamente, constituímos, numa empresa escolhida, dois lotes de 30 trabalhadores que se distinguiam pelo facto de um ter gengivorragias e o outro não.

Sobre cada um destes dois lotes foram feitas as mesmas pesquisas:

1.º Prova da saturação para o ácido ascórbico com 2,6-diclorofenol-indofenol.

2.º Inquérito alimentar quantitativo.

3.º Estabelecimento dos índices malariométricos (parasitário, esplénico, baço-médio), de infestação por bilharziose e por vermes intestinais.

4.º Classificação do género de trabalho de cada um dos indivíduos em ligeiro, médio e pesado.

5.º Além disso, aos portadores de gengivorragias foram administrados 200 mg diários de Redoxon Roche por via oral, durante 45 dias, observando-se periòdicamente o grau das gengivorragias, que, simbòlicamente, eram assim classificadas:

— *ausentes.*
+ *ligeiras.*
++ *médias.*
+++ *acentuadas.*

Com esta última prática e as duas primeiras determinações tínhamos em vista pôr em destaque o papel determinante da vitamina C nas gengivorragias.

Com as restantes pretendíamos achar, pelas variações individuais dos mesmos factores afectando a totalidade dos indivíduos (endemias locais, regimes alimentar e de trabalho), uma justificação para a variação do fenómeno gengivorragia — sua presença ou sua ausência.

Os resultados destas pesquisas podem ser assim sintetizados e ordenados:

1.º A prova da saturação para o ácido ascórbico é positiva na quase totalidade dos indivíduos, quer tenham gengivorragias (97 %) quer não (100 %). A diferença entre estas duas percentagens não é significativa.

2.º Não há diferenças significativas entre os dois grupos, que se possam atribuir a variações individuais, quanto às doenças endémicas que grassavam no local do trabalho (quadro 1).

3.º Tal diferença é significativa quanto ao índice de baço-médio. Os indígenas com gengivorragias apresentavam esplenomegalias maiores (quadro 2).

4.º Não é possível valorizar a influência dos géneros do trabalho em virtude de a distribuição deste não ser feita ao acaso. Os indígenas eram seleccionados pela empresa, de modo que aos mais robustos eram distribuídos os trabalhos mais pesados.

QUADRO 1 — *Comparação nos dois grupos da incidência das doenças endémicas afectando a totalidade dos indivíduos (variações individuais)*

Casos	Paludismo		Bilharziose	Parasitas intestinais
	Índice esplénico	Índice parasitário	Índice de infestação parasitária	Índice de infestação parasitária
Com gengivorragia	43 %	43 %	10 %	80 %
Sem gengivorragia	40 %	48 %	13 %	73 %
Valores de R.	0,265	0,775	0,334	0,698

$$R = \frac{p^2 - p^1}{\sigma} \text{ para } \sigma = \sqrt{\frac{pq}{n^1} + \frac{pq}{n^2}}$$

Significativas as diferenças quando $R > 2$ para uma probabilidade $P = 0{,}05$

QUADRO 2 — *Gengivorragias e baço médio*

Casos	Baços 0	Baços P I P e I	Baços II e III	Total
Com gengivorragia	17	4	9	30
Sem gengivorragia	18	9	3	30
Totais	35	13	12	60

Houve que juntar os P I P e I, II e III para não obter números teóricos inferiores a 5 em 20 $^0/_0$

$X^2 = 2{,}9$ Diferença significativa para uma probabilidade $P = 0{,}05$ e graus de liberdade $n = 2$

5.º As gengivorragias não curaram pela administração da vitamina C. Pelo contrário, o teste do χ^2 evidência a *influência favorável da não administração da vitamina C* (quadro 3).

QUADRO 3 — *Variação das gengivorragias sob a acção da vitamina C (durante 45 dias)*

Estado inicial		Estado final	
No lote com vitamina C	No lote-testemunha	No lote com vitamina C	No lote-testemunha
++	+	+++	+
+	+	+	—
+	+	+	++
+	+	+	+
++	+	++	++
+	+	—	—
+	+	+	—
+	+	+	++
+	+++	++	+
+	+	++	+
+	+	+++	—
++	+	+	+
+	++	+++	+
+	+	+	+
+	+	—	—
+	+	+	+
++	+	++	+
+	++	+	+
+	+	+	+
+	+	+	+
+	+	—	—
+++	+	++	—
+	++	+	—
+	++	—	—
++	++	+	++
+	++	+++	+
+	++	+	—
+	+	+	++
++	+	+	+
++	++	+	+

Casos	Mesmo estado	Melhorados	Curados	Piorados	Total
Com vitamina C . . .	15	5	4	6	30
Sem vitamina C . . .	11	5	10	4	30
Totais . . .	26	10	14	10	60

$X^2 = 3{,}20$ Diferença significativa para uma probabilidade
$P = 0{,}05$ e graus de liberdade $n = 3$

6.º A composição nutritiva das rações difere nos dois lotes. A dos com gengivorragias é mais rica em geral e, em particular, em vitamina C (quadro 4).

QUADRO 4 — *Composição comparada das rações dos indivíduos com e sem gengivorragias*

Casos	Calorias	Proteinas		Lipidos (gramas)	HCO (gramas)	Cálcio (miligramas)	Ferro (miligramas)	Vitamina A (U. I.)	Tiamina (miligramas)	Riboflavina (miligramas)	Niacina (miligramas)	Ácido ascórbico (miligramas)
		Animais (gramas)	Vegetais (gramas)									
Com gengivorragia . . .	3 716	94	84	93	540	3 759	51,19	88	2,46	1,55	25,31	2,50
Sem gengivorragia . . .	2 248	56	56	50	340	2 208	35,32	12	1,88	1,16	12,67	0,38

Valores obtidos por inquérito directo, por pesagem, com amostragem na fracção 1/5

Este facto concorda com o citado no número anterior.

O conjunto dos resultados referidos no n.º 1.º e no n.º 6.º mostra ainda que, a despeito de rações pobres em ácido ascórbico, há uma excreção urinária de vitamina de montante suficiente para dar provas de saturação positivas.

Em 1953 um de nós (4) observou que certos sinais de carência de riboflavina (glossites atróficas) nos trabalhadores indígenas desapareciam pela passagem dos serviçais do regime de trabalho ao de repouso, mantendo-se iguais as restantes condições de vida — dieta, geoclima, etc. Por outro lado, foi também verificado que estas glossites quase nunca se observavam nos indígenas que, oriundos das suas terras, eram inspeccionados antes de seguirem para os locais de trabalho.

Neste último aspecto havia, pois, uma semelhança de situações entre as glossites e as gengivorragias.

Estas, com efeito, como já dissemos, nas inspecções prévias ou não aparecem ou aparecem numa percentagem média que não ultrapassa 3 % a 5 %, ao passo que nos locais de trabalho tal percentagem média atinge 26 %-23 % para as empresas piscatórias, 19 % para as empresas do café e 30 % para as do sisal.

Como do estudo sobre as glossites resultou admitir-se uma carência secundária, por aumento de excreção urinária de riboflavina, consecutiva ao *stress* a que os trabalhadores eram sujeitos ao virem para os locais de trabalho, considerou-se que esta hipótese seria sedutora para explicar as gengivorragias que não curavam a despeito da administração de vitamina C.

Realmente não era só a diferença de percentagens das gengivorragias nas inspecções prévias e nos locais de trabalho que nos levavam a pensar assim.

Nós próprios verificávamos que, a despeito de baixas ingestas de vitamina C, a sua eliminação pela urina se continuava fazendo em quantidades tais que a prova da saturação era positiva. E, por outro lado, sabíamos como muitos «stressores» provocam o aumento da excreção urinária da vitamina. Acontece assim, por exemplo, com o frio — Thérien e Dugal (5) —, os traumatismos, as queimaduras e o exercício muscular intenso — Delachaux (6).

Que os indígenas sofrem as condições de um marcado *stress* quando mudam do seu *habitat* usual, do meio rural livre, para os locais de trabalho, não pode oferecer dúvidas.

Além das variações das condições geoclimáticas, às vezes tão acentuadas como as diferenças entre as frescas e secas matas do planalto e o deserto quente e batido pelos ventos do litoral, além das condições alimentares, onde uma alimentação livre e de recursos múltiplos dá lugar a refeições monòtonamente standardizadas, há o trauma psicológico resultante do afastamento do meio habitual e, sobretudo, uma modificação completa no regime de actividade muscular. Aqui a doce ociosidade, apenas cortada pelas práticas da pesca, da caça e da conservação da cubata, porque é a mulher que faz todo o resto, dá lugar à tarefa que se tem de cumprir sob o olhar vigilante de capatazes pouco dados a condescendências.

Este *stress* provocará evidentemente, como sempre, a resposta endócrina pela qual o organismo se procura adaptar à nova situação, através de uma secreção aumentada de hormonas de adaptação, em especial a ACTH e a cortisona.

Se assim fosse, nos nossos casos era de prever que a aplicação destas hormonas modificasse, pela modificação da excreção da vitamina, as gengivorragias. Concretizando: se houvesse deficiência de hormonas, hipoadaptação, a sua aplicação, aumentando a eliminação urinária da vitamina, faria aparecer as gengivorragias nos casos em que não existissem ou aumentaria a intensidade das existentes.

No caso contrário, hiperadaptação, a única modificação notável que se poderia apreciar seria o seu aumento.

Com o objectivo de esclarecer esta alternativa, constituímos 5 lotes de 8 trabalhadores cada:

LOTE I — Indivíduos com gengivorragias aos quais administrávamos a cortisona.
LOTE II — Com gengivorragias administrando ACTH.
LOTE III — Sem gengivorragias administrando cortisona.
LOTE IV — Sem gengivorragias administrando ACTH.
LOTE V — Com gengivorragias-testemunhas.

Ao mesmo tempo estudávamos a eliminação urinária de ácido ascórbico.

Nos quadros 5, 6 e 7 estão sintetizados os resultados obtidos depois de se terem aumentado os lotes I, II e V para 30 casos cada.

Como se poderá observar, nenhum dos resultados supostos se verificou. Pelo contrário, a aplicação das hormonas curaram as gengivorragias nos que as tinham, não exercendo qualquer efeito sobre os restantes.

Ao aceitarmos a possibilidade de as hormonas do *stress* desencadearem ou aumentarem as gengivorragias partíamos do princípio de que o fariam através de uma carência secundária de vitamina C.

Como o estudo da eliminação urinária desta vitamina por acção das hormonas mostrou que, após um transitório aumento, tal eliminação diminui e como as ingestas se mantêm na mesma, não há lugar para admitir uma carência secundária por sobreeliminação.

O efeito curativo, mais nítido com a cortisona do que com o ACTH, só se pode interpretar correctamente admitindo que a acção das hormonas, neste caso, qualquer que ela seja, se exerce sem interferência da vitamina C. Assim, mais uma vez, e por vias diferentes, se confirma o que já havíamos admitido como resultado das observações clínicas: não há entre o escorbuto e estas gengivorragias qualquer relação de dependência.

Como interpretar então a acção curativa das hormonas?

QUADRO 5 — *Comportamento das gengivorragias pela aplicação da ACTH*

Com ACTH						Testemunha		
Com gengivorragias			Sem gengivorragia			Com gengivorragias e sem hormonas		
Estado inicial	Estado final (9.º dia)	Desaparecidas no dia	Estado inicial	Estado final (9.º dia)	Aparecidas no dia	Estado inicial	Estado final (9.º dia)	Desaparecidas no dia
+	—	4	—	+	2	++	—	4
+	—	4	—	—	—	++	+	—
+	—	6	—	—	—	++	++	—
++	+	—	—	—	—	+	+	—
+	—	2	—	—	—	+	+	—
+	—	2	—	—	—	++	+	—
+	—	4	—	—	—	+	—	2
+	—	5	—	—	—	++	+	—
+	+	—				+	++	—
++	—	4				+	+	—
++	++	—				+	—	3
+	—	2				+	—	5
+	+	—				+	+	—
+	+	—				+	+	—
+	—	4				+	—	2
+	—	2				+++	++	—
++	+	—				++	+	—
+	+	—				+	+	—
++	++	—				+	—	2
++	+	—				++	+	—
++	+	—				+	+	—
++	+	—				++	+	—
++	+	—				+	—	2
+	+	—				+++	++	—
++	—	3				+	—	2
++	+	—				+++	++	—
+	—	5				+	—	2
++	—	6				+	+	—
++	+	—				++	+	—
+	+	—				+	—	7

Injecção intramuscular diária de *Cortrophine — Z «Organon»* durante 7 dias com a seguinte pauta:

1.º dia: 30 U. I. — 1,5 cm^3

2.º e 3.º dias: 20 U. I. — 1 cm^3

4.º e 5.º dias: 15 U. I. — 0,75 cm^3

6.º e 7.º dias: 10 U. I. — 0,50 cm^3

QUADRO 6 — *Comportamento das gengivorragias pela aplicação da cortisona*

Com cortisona						Testemunha		
Com gengivorragias			Sem gengivorragias			Com gengivorragias e sem hormonas		
Estado inicial	Estado final (9.º dia)	Desaparecidas no dia	Estado inicial	Estado final (9.º dia)	Aparecidas no dia	Estado inicia	Estado final (9.º dia)	Desaparecidas no dia
+	—	3	—	+	3	++	—	4
+	—	3	—	—	—	++	+	—
++	—	2	—	—	—	++	++	—
+	—	2	—	—	—	+	+	—
+	—	2	—	—	—	+	+	—
+	—	2	—	—	—	++	+	—
+	—	2	—	—	—	+	—	2
+	+	—	—	—	—	++	+	—
++	—	4	*Adreson «Organon»* por via oral durante 7 dias, em comprimidos doseados a 25 mg, com a seguinte pauta: 1.º dia: 300 mg — 4 cm³ de 8 em 8 horas 2.º dia e restantes: 150 mg — 2 cm³ de 8 em 8 horas			+	++	—
+	—	2				+	+	—
+	—	3				+	—	3
+	—	4				+	—	5
++	+	—				+	+	—
+	—	2				+	+	—
+	—	4				+	—	2
+	—	5				+++	++	—
+	—	6				++	+	—
+	—	3				+	+	—
++	+	—				+	—	2
+	—	2				++	+	—
++	—	3				+	+	—
+	—	2				++	+	—
+	+	—				+	—	2
+	—	2				+++	++	—
++	—	2				+	—	2
+	—	3				+++	++	—
+	—	2				+	—	2
+	+	—				+	+	—
+	—	2				++	+	—
+	—	2				+	—	7

QUADRO 7 (resumo dos quadros 5 e 6) — *Comportamento das gengivorragias pela aplicação das hormonas de adaptação*

Com cortisona				Com ACTH				Testemunha sem hormonas			
Curados	Não curados	Percentagem de curas	Prazo médio de cura (dias)	Curados	Não curados	Percentagem de curas	Prazo médio de cura (dias)	Curados	Não curados	Percentagem de curas	Prazo médio de cura (dias)
25	5	83%	2,76	14	16	47 %	4,79	10	20	33 %	4,10

A análise estatística destes resultados mostra:

1.º Curas devidas à cortisona comparadas com a testemunha:
$R = 3{,}984$ — significativa para $P = 0{,}05$

2.º Curas devidas ao ACTH comparadas à testemunha:
$R = 2{,}077$ — significativa para $P = 0{,}05$

3.º Curas devidas à cortisona comparadas a ACTH:
$R = 3{,}124$ — significativa para $P = 0{,}05$

$$R = \frac{p^2 - p^1}{\sigma} \text{ para } \sigma = \sqrt{\frac{pq}{n^1} + \frac{pq}{n^2}}$$

Significativas as diferenças quando $R > 2$ para uma probabilidade $P = 0{,}05$

Com a aplicação das hormonas a excreção urinária da vitamina segue um curso bifásico, quer haja gengivorragias quer não (gráfico I).

A ascorbinúria aumenta a partir do dia imediato ao da aplicação, para diminuir bruscamente a partir do quarto ou do quinto dia. Esta diminuição, numa grande parte dos casos, é tão brusca que 45 cm^3 de urina não descoram o soluto com 2 mg de clorofenol indofenol. Enquanto no dia anterior menos que a quarta parte desse volume o fazia.

Noutros casos a ascorbinúria, que primeiro havia aumentado, começa diminuindo e em dois ou três dias desaparece ou, melhor, atinge um quantitativo tal que 45 cm^3 não descoram o soluto indicador.

Como se verifica no gráfico, o traçado destas linhas é o mesmo tanto nos indivíduos com gengivorragias como nos que as não têm, o que constitui mais um argumento para não aceitar qualquer relação entre o fenómeno e a excreção urinária da vitamina C.

São contraditórios os elementos que se encontram na literatura sobre a excreção urinária de vitamina C pela acção das hormonas de adaptação. Stefanini e Rosenthal (7) e Booker (8) verificaram uma excreção diminuída; Beck e col. (9), pelo contrário, verificaram um aumento; Bicknell e Prescott (10) resumem que alguns observadores não referem modificações significativas, ao passo que outros mencionam uma excreção aumentada.

Não encontrámos referência, na literatura disponível, à evolução bifásica por nós verificada.

No entanto, segundo Andreae e Browne (11), a tolerância ao ácido ascórbico aumenta durante o *stress*, ao passo que segundo Booker e col. (12) diminui.

Estudando a ascorbinemia em indivíduos sujeitos a diversos «stressores», Fortier e Skelton (13) verificaram que esta segue uma evolução trifásica, diminuindo primeiro para aumentar depois e, finalmente, voltar a diminuir. A duração das diversas fases depende da gravidade do *stress* e da capacidade de adaptação dos organismos.

Estas variações da ascorbinemia dependem da mobilização da vitamina de certas células (fígado, pulmões, coração, encéfalo) para outras (principalmente supra-renais, mas também os rins e algumas vezes o baço), como as análises histoquímicas de Long (14), Frommel e Beck (15) e outros mostraram.

Quanto aos nossos casos, a variação da ascorbinúria só pode ser interpretada de dois modos: ou pela variação do limiar de excreção renal, ou pela variação da ascorbinemia. Esta, com efeito, será maior ou menor, para as mesmas ingestas, conforme as necessidades de consumo ou de fixação da vitamina nos tecidos sejam menores ou maiores.

A primeira interpretação parece-nos pouco realista, já que implica que a mesma causa, a aplicação da hormona, tenha sobre o mesmo factor, o limiar de excreção renal, dois efeitos opostos.

Sendo a segunda interpretação mais consentânea com a realidade dos factos, incluindo a variação trifásica de Fortier e Skelton, era de esperar que a administração de doses maciças de vitamina C, simultâneamente com as hormonas, ocasionasse a persistência de uma ascorbinúria elevada, isto é, suprimisse a sua brusca queda, tão evidente no gráfico I.

Para confirmar esta presunção e poder fazer a análise estatística dos resultados, além do já citado aumento dos lotes I, II e V com mais

22 casos cada, promovemos que em 18 destes casos (6 de cada lote) se administrasse 1 g diário de ácido ascórbico.

Os resultados obtidos e a sua análise estão juntos aos do ensaio-sondagem que referimos. A estes houve que acrescentar, além da evolução dos novos 66 casos e apreciação estatística final, os dados respeitantes à eliminação urinária de vitamina nos indivíduos em regime de sobrecarga (gráfico II).

Realmente verifica-se que com tal sobrecarga a diminuição da ascorbinúria provocada pelas hormonas deixa de se verificar.

Tudo se passa pois como se os nossos indivíduos se encontrassem em estado de hipoadaptação, com a secreção de hormonas do *stress* abaixo do nível necessário. Em consequência deste baixo nível, os tecidos não fixavam grandes quantidades de ácido ascórbico, o que permitia manter, a despeito das baixas ingestas, uma ascorbinemia e uma ascorbinúria suficientemente elevadas e persistentes.

A administração de hormonas, suprimindo o estado de hipoadaptação, levava à fixação da vitamina pelos tecidos, baixa da ascorbinemia e, portanto, da ascorbinúria.

Entre as duas fases havia uma curta e transitória de ascorbinemia mais elevada, e portanto ascorbinúria mais intensa, correspondente à mobilização da vitamina.

Outra interpretação possível, em princípio, era justificar a baixa da ascorbinúria pela espoliação do organismo em ácido ascórbico, resultante do aumento da excreção urinária deste causado pelas hormonas.

Tal interpretação não é, no entanto, corroborada pelo facto de a ascorbinúria manter um nível muito mais alto quando se faz a sobrecarga sem aplicar as hormonas. Foi o que se verificou em 6 dos nossos casos, cujo resultado conjunto apresentamos no gráfico II.

Com efeito, se para a mesma sobrecarga de vitamina, e partindo de níveis idênticos, a ascorbinúria é muito menor quando se aplicam as hormonas, cabe admitir que a vitamina seja retida, fixada ou consumida, no organismo.

Como interpretar então a acção curativa da cortisona e do ACTH?

Pode-se admitir que se trata de uma acção inespecífica sobre o tecido mesenquimatoso, similarmente ao que para o verdadeiro escorbuto Schaffenburg, Masson e Corcoran (16) descreveram.

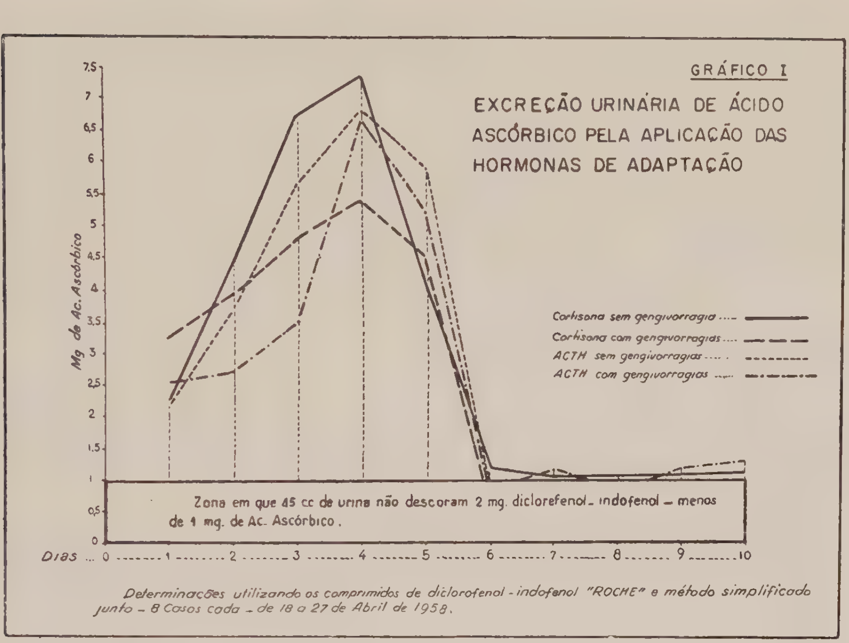

GRÁFICO I

EXCREÇÃO URINÁRIA DE ÁCIDO ASCÓRBICO PELA APLICAÇÃO DAS HORMONAS DE ADAPTAÇÃO

Zona em que 45 cc de urina não descoram 2 mg. diclorefenol – indofenol – menos de 1 mg. de Ac. Ascórbico.

Determinacões utilizando os comprimidos de diclorofenol - indofenol "ROCHE" e método simplificado junto – 8 Casos cada – de 18 a 27 de Abril de 1958.

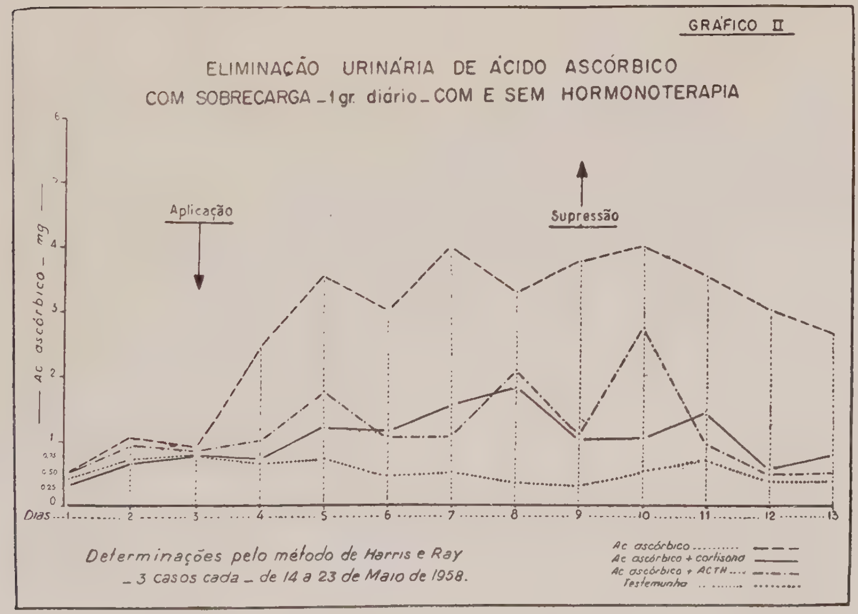

GRÁFICO II

ELIMINAÇÃO URINÁRIA DE ÁCIDO ASCÓRBICO COM SOBRECARGA – 1 gr. diário – COM E SEM HORMONOTERAPIA

Determinações pelo método de Harris e Ray – 3 casos cada – de 14 a 23 de Maio de 1958.

No entanto, o termos verificado que as gengivorragias curam quando os indígenas regressam às suas terras de origem, isto é, quando desaparecem as condições de *stress*, induz-nos a pensar que, realmente, a acção das hormonas resulta da correcção de uma hipoadaptação dos indivíduos a esse *stress*. À mesma conclusão nos leva o facto oposto, isto é, o já citado aparecimento das gengivorragias quando os indígenas saem das suas terras para os locais de trabalho.

Já referimos as condições de *stress* que afectam os indivíduos quando estes se deslocam do seu *habitat* livre para as empresas em regime de trabalho organizado.

Já citámos também os diversos «stressores» que em tais circunstâncias se podem considerar como actuantes. Evidentemente que o total da acção exercida depende do somatório desses «stressores», cujo valor parcelar exacto será diferente em cada caso.

Parece-nos, no entanto, que o trabalho muscular intensivo é, no caso dos trabalhadores indígenas, entre todos, o que mais se avoluma.

Os efeitos do exercício muscular intenso como o «stressor» foram descritos por vários autores, quer nos animais — Selye (17), Ratsimananga (18) —, quer no homem — Venning e Kasmin (19) —, e como consequência Selye (20) estabelece o conceito de que tais efeitos resultam de uma hipoadaptação.

O efeito curativo das hormonas de adaptação sobre as gengivorragias e a maneira como estas se comportam quando os indígenas mudam do seu *habitat* natural para os locais de trabalho e vice-versa, bem como os resultados das investigações em que pretendemos relacionar este fenómeno com a carência de vitamina C, com as condições alimentares e endémicas existentes nesses locais, levam-nos a admitir que as gengivorragias são um sintoma pelo qual se manifesta a hipoadaptação dos indivíduos.

Tal hipoadaptação deve resultar, como habitualmente, de uma diminuída capacidade de resposta das supra-renais aos estímulos criados pelas condições de *stress*. Por isto mesmo é que uma terapêutica substitutiva da secreção supra-renal (cortisona) deve ter um efeito curativo muito mais nítido e em prazo muito mais curto que uma terapêutica estimulante da glândula (ACTH).

Esta hipótese, que a nossa teoria permitiu formular, corresponde precisamente aos factos verificados (quadro 7).

Evidentemente que nesta hipoadaptação, como sempre, intervêm, por um lado, a capacidade de adaptação dos organismos, que é variável, e, por outro, o somatório, também variável, dos «stressores» que actuam.

Precisamente por esta variabilidade é que, como tantas vezes observámos, a evolução das gengivorragias é caprichosa, desaparecendo e reaparecendo espontâneamente, o que nos obrigou a uma análise estatística cuidadosa, comparando com testemunhas, antes de concluirmos sobre o efeito curativo das hormonas.

Dos «stressores» o que *a priori* parece ser o mais importante é o trabalho muscular intenso.

Actualmente estão em curso investigações destinadas a averiguar o seu valor e, conjunta e complementarmente, dos restantes.

RESUMO

Os AA. descrevem uma nova entidade mórbida — as gengivorragias que se verificaram nos trabalhadores indígenas de Angola como sintoma de um síndroma geral de adaptação (SGA).

Descrevem a técnica que utilizaram para a sua identificação e as investigações a que procederam para esclarecer a etiologia do processo.

Como consequências dessas investigações, os AA. verificaram:

1.° Que as gengivorragias não são devidas, como era geralmente admitido, a uma carência de vitamina C. Com efeito:

a) Apesar de uma baixa percentagem de adequação desta vitamina revelada no inquérito alimentar quantitativo, os indivíduos eliminam vitamina C pela urina de modo idêntico, quer tenham gengivorragias quer não;

b) A administração de 200 mg diários de vitamina C, durante 45 dias, não cura as gengivorragias em percentagem significativa.

2.° Que estas gengivorragias não se verificam quando os indivíduos são observados nas inspecções que precedem o seu deslocamento para os locais de trabalho.

3.° Que, uma vez chegados a estes locais, as gengivorragias aparecem sob forma «epidémica».

4.° Que em percentagem variável se mantêm durante a totalidade de permanência dos indivíduos nos locais de trabalho.

5.° Que, uma vez regressado ao seu *habitat* de rural, as gengivorragias desaparecem.

6.º Que as gengivorragias não têm qualquer relação significativa com as moléstias endémicas que atacam estes indivíduos nos locais de trabalho — paludismo, bilharziose, verminoses intestinais.

Perante estes resultados, os AA. admitem como causa provável a influência do *stress* a que os indígenas são sujeitos ao serem deslocados da sua vida livre para um regime de trabalho organizado em regiões diferentes do seu *habitat*.

De acordo com esta hipótese, fazem a aplicação das hormonas do *stress* (cortisona e ACTH) e verificam que, tanto num caso como noutro, se obtém um efeito curativo nítido sobre as gengivorragias.

A fim de explicar a eliminação de ácido ascórbico pela urina em indivíduos cujas rações são deficientes na vitamina C, os AA. estudaram a influência daquelas hormonas sobre esta eliminação e verificaram que, realmente, esta diminui após um aumento transitório.

Estes factos não só permitem compreender o resultado contraditório de uma eliminação urinária de vitamina C com ingestas insuficientes, mas constituem ainda um argumento a favor da intervenção das hormonas do *stress* no quadro de que as gengivorragias fazem parte.

Finalmente, os AA. fazem a revisão das possíveis causas deste *stress* e admitem que o trabalho muscular activo é o mais provável. Estudos destinados a confirmar esta hipótese estão actualmente em curso.

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GENERAL DISCUSSION

Dr. E. M. Glaser (U.K.) — I was very interested in the remark of Commander Minard that special arrangements were made for the obese, and I am wondering whether he or any other speaker have any comments to make on the problem of added heat stress for people who are very fat.

Dr. W. V. Macfarlane (U.S.A.) — The polyuria of anhidrotic heat exhaustion could be primarily a polydipsia occurring after long exposure to heat. In sheep we found that 6 months at 42°C for plus a day produced great (30 e./day) polydipsia. Yet the plasma antidiuretic substance reached 300 μU/ml, a very high level, while urine flowed at 15-20 ml/min. Recovery was rapid at 25°C. The plasma electrolytes were diluted during the polydipsia and this could account for the weakness and blood pressure changes.

It may be that anhidrosis is incidental in man to a heat induced polydipsia, with high ADH output. Adrenal steroid (17 KGS) excretion is reduced at once during heat in man. This does not suggest that the adrenal would be exhausted.

Dr. G. Lambert (France) — L'établissement de l'index de charge thermique dont a parlé le Com. Minard ne mentionne pas la mesure directe de la vélocité de l'air.

Est-ce parce que la valeur due au globe thermomètre est suffisante pour apprécier cette vélocité?

Réponse du Com. Minard: Oui, dans les conditions d'utilisation de l'index.

Commander David Minard (U.S.A.) — *Response to Dr. Glaser:* Special protection is given obese recruits because the association between obesity and susceptibility to heat stroke has been demonstrated by Elizabeth Schickele in army recruits in WW II and in more recent studies in the U.S. Army. Whether body habitus is the only

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factor in this relationship is doubtful. Obese recruits at Parris Island are returned to regular training units following a period of supervised training and weight reduction on a low caloric diet. *Response to Dr. Lambert:* The WBGT index does include all four physical variables. The globe, in effect, acts as an anemometer in the presence of a positive radiation load. As Prof. Belding pointed out, there is no universal index of heat stress. Although Prof. Yaglou has used the WBGT successfully at Yuma, Arizona, it may be that an index which includes actual measurement of wind velocity would be better suited for conditions existing in the Sahara desert. *Response to Prof. Crowden:* The Bedford Corrected Effective Temperature gives as good a correlation coefficient with our data as does the WBGT index. However, the instruments used in determining the WBGT index are simple and can be maintained and read by non-technical personnel. The Bedford C.E.T. requires experienced technical personnel in obtaining measurement of air movement by Kata-thermometer.

SURGEON COMMANDER F. P. ELLIS (England) — I would like to support Air Commodore Stamm's suggestion that the skin disorders and neuropsychiatric conditions should be excluded from the Section of the International Classification of Diseases which covers the incapacitating heat illnesses and be included in the appropriate dermatological and psychiatric sections elsewhere, and in addition his proposal that heat cramps be included under heat exhaustion.

At the same time, further consideration might be given to the nomenclature and nature of these two groups of heat disorders before the next International Congress meets and the programme could then be expanded to include more time for discussion of the effects of sun on the skin and of the miliarias, and also of the psychiatric and psychological states associated with living and working in hot climates. Additional time could also be devoted with great benefit to salt and water requirements in hot humid and dry climates, a topic of great practical importance which has only been touched on superficially at this Congress.

A short time ago I received a communication from Dr. K. Cameron who reviewed the psychiatric aspects of tropical service in the Royal Navy in the *Journal of Mental Science* in 1949. With reference to the term «heat neurotic reaction» he was doubtful of the wisdom of applying the description «neurotic» to men essentially of good personality who were temporarily exhibiting neurotic signs and symptoms because of exposure to unduly severe or prolonged heat stress, and suggested that the term «tropical (or hot-climate) personality reaction», mild or severe, be used, to which the terms «with neurosis» or «with psychosis» could be added in cases where the clinical features merited this (Slide 1).

SLIDE 1 — *Clinical classification of «tropical» neuro-psychotic disorders (after Cameron, 1958, personal communication)*

- Tropical personality reaction . .
  - Mild
  - Severe
  - With neurosis
    - Hysterical
    - Anxiety
    - Other
  - With psychosis
    - Depressive
    - Manic
    - Confusional

It has recently been suggested to Medical Officers in the Royal Navy that the following abbreviated list will cover the great majority of incapacitating heat illnesses encountered in the naval service (Slide 2).

SLIDE 2 — *Incapacitating heat illness — Classification proposed for trial in the Royal Navy (Ellis, 1958)*

I.S.C.[1] No. (1957)	
N.981.0	Heat hyperpyrexia (heat stroke)
N.981.3	Heat exhaustion (salt and/or water deficiency states, including heat cramps)
N.981.3	Anhidrotic heat exhaustion
N.981.3	Heat syncope (sudden loss of consciousness with changes in posture, whilst standing erect for a prolonged period on parade, or on entering an excessively warm atmosphere to which one is not accustomed)
318.3	Hot-climate fatigue (excessive fatigue in the absence of notable thermoregulatory failure, salt or water deficiency)

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[1] I.S.C. (1957) = International Statistical Classification — W.H.O. (1957).

The term «hot-climate fatigue» or «tropical fatigue» is preferred to Cameron's «hot-climate personality reaction». The artificial distinction between heat stroke and heat hyperpyrexia is eliminated and the latter term only is retained.

It was a surprise to me after reading Dr. Ladell's paper on anhidrotic heat exhaustion that such a clearly defined condition was so rarely reported in the British Navy in the Tropics in World War II, where prickly heat was rife and acute heat incapacitation a not uncommon event amongst the ship's companies who were continuously exposed to very hot, humid conditions for many months on end. In a recent series of over 2,000 cases of heat illness in the United States Army, Cook (1955) observed only 20 cases of heat stroke and he did not mention anhidrotic heat exhaustion as such. Was this because the duration of exposure to heat was relatively brief in Cook's series? Can this dearth of naval cases of anhidrotic heat exhaustion have been due to failure to recognise this type of case or can it be attributed to the fact that, although exposure to humid heat was often prolonged, it was not usually associated with prolonged exposure to solar radiation, a fact which perhaps distinguishes the naval experience afloat from that of the Army and Royal Air Force troops on shore? There may be an aetiological clue here which could profitably be followed up!

SUBSECTION 2

NUTRITION

1ST SESSION: **Malnutrition: some outstanding public health problems**

Wednesday, September 10th — 9:00 to 10:30 a.m. Room B

The meeting was held under the chairmanship of Dr. B. S. Platt.

The Rapporteur was Dr. J. C. Waterlow and the Secretary was Dr. Carlos dos Santos Reis.

The following papers were presented and discussed.

THE COMPOSITION OF MILK FROM AFRICAN MOTHERS IN THE KIVU REGION OF THE BELGIAN CONGO

O. A. ROELS [1]
and
M. TROUT
Laboratoire de Nutrition
Institut pour la Recherche Scientifique en Afrique Centrale
Lwiro (Kivu), Congo Belge

Maternal milk is a very important part of the diet of young children in Tropical Africa. Its quantity and composition are therefore of great interest to nutritionists and paediatricians alike, because these factors influence growth and health of the children for a fairly long period, since suckling is normally continued for well over a year and sometimes for up to 3 years. Moreover, only a small percentage of the mothers in these regions have animal milk or other high quality foods available for the baby as a supplement or a substitute for their own milk.

It was necessary to obtain precise qualitative and quantitative data about the milk secreted by African mothers to understand more about prevalent nutritional deficiencies and their prevention among young children (Brock and Autret. 1952). We have therefore undertaken a study of the chemical composition of milk from African mothers living in the Kivu region of the Belgian Congo.

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[1] Biochemist in the Medical Department of the Belgian Congo Government.

The milk samples were collected by Dr. E. Demaeyer of this department, who has undertaken a study (to be published) of the amount of milk secreted per 24 hours by African mothers living in their customary rural environment near our Laboratories. All the milk donors belong to the Bashi tribe. This group lives along the Eastern shore of Lake Kivu, in the Belgian Congo (about 29° East of Greenwich), between about 2° 10′ South and 2° 40′ South, at an altitude varying from 1,460 m to 2.000 m above sea level.

The ages of the mothers ranged from 17 to 40 years. Their staple foods are beans, sweet potatoes and beer made from bananas.

In comparing our results with those of previous workers, we have mainly used the important study of human milk made by Kon and Mawson (1950) in England between 1941 and 1945 on over 2,000 samples of milk. Some of our analytical methods where inspired by their work. We have also used the extensive literature surveys done by Morrison (1952), by Macy, Kelly and Sloan (1953) and by Janz, Demaeyer and Close (1957), who list and summarize most of the individual publications in this field.

METHODS

All the determinations except those of vitamin A and carotene were done in duplicate. No colostrum samples are included in this work.

1. *Collection of milk from the donors* (Demaeyer, to be published. — The mothers remained in the dispensary of our Institute for a period of 24 hours or longer. The milk samples used for the analyses were collected over a 24 hour period by manual extraction of both breasts at regular intervals. The babies were separated from the mothers and bottle-fed for that period. The milk was kept under refrigeration during the period of collection.

2. *Preparation of homogeneous samples.* — The milk collected over the 24 hour period was brought to 20°C and mixed until homogeneous by pouring it to and from beakers. If this procedure did not disperse large globules of fat, then the sample was warmed to 38°C on a water bath and the same mixing procedure was repeated at this temperature (A.O.A.C., 1955). The samples for the various determinations were weighed and the analyses were started on the day immediately following the day of collection.
3. *The total solids* were determined according to Method I of the Association of Official Agricultural Chemists (A.O.A.C., 1955, p. 246).
4. *The ash* was determined by calcinating the «total solids» at 550°C to constant weight (A.O.A.C., 1955, p. 248).
5. *The fats* were determined by the Roese-Gottlieb method (A.O.A.C., 1955, p. 248).
6. *The total nitrogen* in the milk was determined by the Macro Kjeldahl method of Hiller, Plazin and Van Slijke (1948).
7. *The lactose content* of the milk was determined by a method developed in the National Institute for Research in Dairying (Reading, England) from a procedure described earlier by Hinton and Macara (1927).
8. *The Vitamin A and carotene* determinations in the milk were done according to Henry and Thompson (1954).

It should be noted that all our results are expressed in grams per 100 g of milk. Since most literature reports on the composition of human milk are given in grams per 100 ml, our results should be increased by about 3 % to make them comparable to those given per 100 ml. Indeed, Macy (1953) reports the average density of human milk to be 1.031, and Holemans, Lambrechts and Martin (1954) give 1.030 for the Kwango region of the Belgian Congo.

THE TOTAL NITROGEN AND THE «PROTEINS» OF THE MILK

Table 1 gives the variation of the total nitrogen in milk (excluding colostrum) with the stage of lactation:

TABLE 1 — *Variation of total nitrogen with stage of lactation*

Month	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
1st	20	0.229	0.033	0.170	0.310
2nd	27	0.206	0.040	0.147	0.317
3rd	28	0.180	0.028	0.134	0.232
4th	22	0.173	0.027	0.098	0.211
5th	19	0.178	0.034	0.134	0.267
6th	20	0.164	0.019	0.139	0.218
7th	15	0.171	0.020	0.140	0.201
8th	13	0.156	0.018	0.122	0.184
9th	11	0.157	0.023	0.109	0.183
10th	9	0.148	0.019	0.109	0.172
11th	1	0.173	—	—	—
12th	6	0.199	0.043	0.147	0.279
13th	6	0.163	0.033	0.131	0.229
14th	5	0.173	0.024	0.143	0.214
15th	8	0.161	0.016	0.144	0.195
16th	3	0.162	0.069	0.129	0.196
17th	5	0.162	0.018	0.155	0.173
18th	1	0.135	—	—	—
19th	1	0.163	—	—	—
20th	1	0.141	—	—	—
22nd	3	0.161	0.018	0.137	0.178
31st	1	0.261	—	—	—
32nd	1	0.177	—	—	—
Total	226	0.179	—	0.098	0.317

There is a fairly rapid decrease during the first 3 months after birth. As from the 4th month, the nitrogen content seems to remain more or less constant with perhaps a slight further decrease. Thus we found the mean nitrogen content of the milk secreted during the 1st month (0.229 g/100 g of milk) to be significantly higher ($P < .05$) than that of the milk secreted in the 2nd month (0.206 g/100 g of milk) and the latter was significantly higher ($P < .01$) than that of the milk secreted during the 3rd month (0.180 g/100 g of milk). The difference between the mean nitrogen content of the milk secreted during the 3rd month and that of the 4th month was not statistically significant. Similar observations were made by Kon and Mawson (1950) in England, by Close (1957) in this region and by several other authors, whose reports were discussed in detail by Morrison (1952).

We found the mean value for the total nitrogen in the milk secreted after the 3rd month to be 0.167 g/100 g for the mothers in this region.

This is lower than the 0.187 g of nitrogen per 100 ml of milk found by Kon and Mawson (1950) for English mothers in the period between the 13th and the 28th week of lactation. Our mean value of 0.179 g of total nitrogen per 100 g of milk for the whole period of lactation (from the 1st until the 32nd month excluding colostrum) is considerably lower than the mean value of 0.227 g nitrogen per 100 ml for the milk secreted after the 10th day which was calculated by Macy, Kelly and Sloan (1953) and was based upon a large number of reports from various parts of the world.

For the conversion of total nitrogen to «proteins», we have used the factor 5.12 calculated by Close (1956) from his results of total nitrogen and amino acid determinations made on milk from African mothers living in the same region as those who gave the samples for the present work.

Table 2 summarizes the variation of the «protein» content of the milk with the stage of lactation.

As this table is based on the results of Table 1, the same remarks are applicable. It should be noted that our results for «protein» (based upon the total nitrogen content of the milk) are not comparable with those of most other authors because the most commonly used factors for converting total nitrogen to proteins are 6.25 and 6.37 where as we used 5.12, which seems more accurate in the light of recent work done in this region.

Tables 3 and 4 summarize respectively the variation of the total nitrogen and of the «protein» content with the volume of the milk secreted per 24 hour period.

These results do not show a definite systematic variation.

Morrison (1952) states that there is no diurnal variation in the total nitrogen or «protein» content of human milk. This is further confirmed by comparing our results with those of Close (1957), who did determinations of total nitrogen in small samples of human milk taken from mothers in this region when they came to a baby clinic.

TABLE 2 — *Variation of «protein» (= nitrogen × 5.12) with stage of lactation*

Month	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
1st	20	1.171	0.169	0.870	1.587
2nd.	27	1.056	0.206	0.753	1.623
3rd	28	0.922	0.143	0.599	1.188
4th	22	0.887	0.140	0.502	1.080
5th	19	0.910	0.172	0.686	1.367
6th	20	0.842	0.095	0.712	1.116
7th	15	0.875	0.103	0.717	1.029
8th	13	0.801	0.092	0.625	0.942
9th	11	0.806	0.119	0.558	0.937
10th	9	0.760	0.096	0.558	0.881
11th	1	0.886	—	—	—
12th	6	1.201	0.218	0.753	1.428
13th	6	0.836	0.167	0.671	1.172
14th	5	0.888	0.123	0.732	1.096
15th	8	0.826	0.083	0.737	0.998
16th	3	0.828	0.141	0.660	1.004
17th	5	0.830	0.035	0.794	0.886
18th	1	0.691	—	—	—
19th	1	0.835	—	—	—
20th	1	0.722	—	—	—
22nd.	3	0.826	0.090	0.701	0.911
31st	1	1.336	—	—	—
32nd.	1	0.906	—	—	—
Total . .	226	0.917	0.184	0.502	1.623

For 247 of these samples of milk from the 4th until the 17th month of lactation, he found an average total nitrogen content of 0.169 g per 100 ml. This is not very different from our result of 0.167 g per 100 g on 151 samples of all the milk secreted in 24 hours at the same stage of lactation.

It would thus appear that after an initial decline of the nitrogen content of the milk during the first three months of lactation, there is little change either with the stage of lactation, with the total volume of milk secreted per 24 hours or in the course of the day, although

TABLE 3 — *Variation of total nitrogen with volume of milk secreted per 24 hour period*

Volume of secretion	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
Between:					
0 ml and 100 ml . . .	6	0.173	0.037	0.122	0.224
100 ml and 200 ml . . .	25	0.170	0.027	0.119	0.226
200 ml and 300 ml . . .	56	0.180	0.040	0.130	0.317
300 ml and 400 ml . . .	46	0.186	0.037	0.133	0.310
400 ml and 500 ml . . .	42	0.180	0.034	0.098	0.255
500 ml and 600 ml . . .	25	0.175	0.039	0.109	0.267
600 ml and 700 ml . . .	14	0.191	0.038	0.142	0.296
700 ml and 800 ml . . .	7	0.180	0.034	0.146	0.249
800 ml and 900 ml . . .	1	0.155	—	—	—
900 ml and 1,000 ml . . .	1	0.155	—	—	—
1,100 ml and 1,200 ml . . .	1	0.160	—	—	—
Total	224	0.180	—	0.098	0.317

TABLE 4 — *Variation of «protein» (nitrogen × 5.12) with volume of milk secreted per 24 hour period*

Volume of secretion	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
Between:					
0 ml and 100 ml . . .	6	0.883	0.190	0.625	1.147
100 ml and 200 ml . . .	25	0.866	0.144	0.558	1.157
200 ml and 300 ml . . .	56	0.923	0.205	0.666	1.623
300 ml and 400 ml . . .	46	0.950	0.191	0.681	1.587
400 ml and 500 ml . . .	42	0.921	0.175	0.502	1.306
500 ml and 600 ml . . .	25	0.895	0.200	0.558	1.367
600 ml and 700 ml . . .	14	0.940	0.123	0.727	1.147
700 ml and 800 ml . . .	7	0.920	0.173	0.748	1.275
800 ml and 900 ml . . .	1	0.794	—	—	—
900 ml and 1,000 ml . . .	1	0.794	—	—	—
1,100 ml and 1,200 ml . . .	1	0.819	—	—	—
Total	224	0.917	0.184	0.502	1.623

the nitrogen content of the milk of mothers having a rather poor diet (as in the case of those in this area) seems to be distinctly lower than that of well fed mothers (e.g., various reports about European and American mothers). Holemans (1954) observed an increase in the nitrogen-content of the milk of mothers in the Kwango region of the Belgian Congo after they were given a daily supplement of 40 g dried skim milk powder.

THE FATS IN THE MILK

Table 5 gives the variation of total fats with the stage of lactation.

There does not appear to be any consistent change in the fat content of the milk from month to month. This is in agreement with the findings of Kon and Mawson (1950) in England. It should however be noted in this connection that we have no colostrum samples included in our results, and previous workers seem to have found some evidence for an increase in the fat content of the colostrum with time.

Table 6 reports the fat content in function of the milk yield per 24 hours.

Here again there does not appear to be a systematic change. Morrison (1952), who studied most previous literature reports on the subject, did not find indications of any gradual variation in the fat content of human milk with volume either.

Our mean fat content of 3.22 g per 100 g of milk is lower than the 3.8 g per 100 ml of milk reported by Macy *et al.* (1953) as a representative summary value for mature human milk based upon most of the literature reports published before 1953. Morrison (1952) searched the literature and found reports on the fat content of 299 samples of milk collected over a 24 hour period. Their average fat content was 3.33 g per 100 ml milk with a standard deviation of 0.57. This is very close to the 3.22 g/100 g (standard deviation 0.92) we found for our 227 twenty four hour samples: indeed, when using Macy's (1953) value for the density of milk (1.031), our 3.22 g/100 g would correspond to 3.32 g/100 ml. In our case, however, the spread of the results around the mean is much greater.

Table 5 — *Variation of total fats with stage of lactation*

Month	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
1st	19	3.30	0.97	1.06	4.86
2nd.	27	3.34	0.96	1.08	4.89
3rd.	28	3.26	0.80	1.40	4.99
4th	22	2.98	0.83	1.44	5.47
5th	20	3.44	0.85	1.59	4.96
6th	20	3.16	0.90	1.48	4.59
7th	15	3.14	1.06	1.08	4.89
8th	14	3.13	0.66	1.88	4.56
9th	11	2.94	0.89	0.88	4.40
10th	10	3.08	0.76	1.88	4.57
11th	1	2.64	—	—	—
12th	6	3.99	1.60	1.73	5.81
13th	6	2.11	0.53	1.34	3.12
14th	5	3.76	0.51	3.18	4.61
15th	8	3.39	0.80	1.86	4.48
16th	3	2.95	0.91	1.98	4.16
17th	5	3.73	0.59	2.33	3.79
18th	1	2.73	—	—	—
19th	1	3.72	—	—	—
20th	1	3.18	—	—	—
22nd.	4	2.52	0.86	1.17	3.51
31st	1	5.66	—	—	—
32nd.	1	4.59	—	—	—
Total . .	229	3.22	0.923	0.88	5.81

One should be very careful in comparing literature reports of the fat content of human milk: the sampling technique may greatly influence the results. It is indeed well known that there is a diurnal variation in the fat content of the milk, and that there is also a considerable variation in the course of one feed. It is thus very difficult to compare our results with those reported by Kon and Mawson (1950) from England: they found considerably higher mean values for the fat content of the milk, but worked on small samples taken in the course of the morning feed only.

TABLE 6 — *Variation of total fats with volume of milk secreted per 24 hour period*

Volume of secretion	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
Between:					
0 ml and 100 ml . . .	9	3.39	1.195	1.17	5.70
100 ml and 200 ml . . .	26	2.98	1.017	1.40	5.81
200 ml and 300 ml . . .	57	3.29	0.960	0.88	5.66
300 ml and 400 ml . . .	46	3.16	0.838	1.08	4.86
400 ml and 500 ml . . .	40	3.27	0.975	1.08	4.99
500 ml and 600 ml . . .	25	3.11	1.095	1.41	5.47
600 ml and 700 ml . . .	14	3.40	0.711	2.33	4.61
700 ml and 800 ml . . .	7	3.45	0.456	2.78	4.26
800 ml and 900 ml . . .	1	2.78	—	—	—
900 ml and 1,000 ml . . .	1	3.43	—	—	—
1,100 ml and 1,200 ml . . .	1	3.27	—	—	—
Total	227	3.22	0.923	0.88	5.81

THE LACTOSE CONTENT OF THE MILK

Tables 7 and 8 give the variation of the (anhydrous) lactose content of the milk with stage of lactation and with volume secreted per 24 hour period.

There is very little variation in the lactose content of the milk either with stage of lactation or with total milk yield per 24 hours.

Our mean value of 7.09 g of lactose per 100 g (corresponding to 7.31 g/100 ml) of milk (excluding colostrum) is higher than that reported by Kon and Mawson (1950) from England, where they found mean values of 6.92 g and 6.94 g per 100 ml of milk in 2 different towns for 427 and 159 samples respectively. Macy (1953) reports an average of 7.00 g lactose per 100 ml mature human milk for most of the values in the literature prior to 1953. Morrison (1952) reports a mean value of 7.23 g per 100 ml of milk for 1,010 samples reported in the literature with a standard deviation of 0.67. The difference between his value and ours is not statistically significant.

TABLE 7 — *Variation of lactose with stage of lactation*

Month	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
1st	15	7.05	0.20	6.50	7.37
2nd.	22	6.94	0.32	6.08	7.38
3rd	25	7.13	0.26	6.44	7.51
4td	18	7.01	0.41	5.84	7.39
5th	18	7.16	0.25	6.54	7.69
6th	16	7.20	0.24	6.70	7.50
7th	14	7.11	0.33	6.54	7.76
8th	9	7.13	0.33	6.41	7.52
9th	9	6.96	0.43	6.20	7.44
10th	8	7.13	0.24	6.83	7.55
11th	1	7.28	—	—	—
12th	6	7.06	0.74	5.55	7.81
13th	5	7.00	0.34	6.42	7.33
14th	5	7.15	0.22	6.86	7.46
15th	8	7.33	0.33	6.62	7.65
16th	2	7.35	—	—	—
17th	4	6.95	0.36	6.60	7.41
18th	1	6.91	—	—	—
19th	1	7.46	—	—	—
20th	1	6.81	—	—	—
22nd.	4	7.08	0.57	6.10	7.50
31st	1	6.81	—	—	—
32nd.	1	7.05	—	—	—
Total. . .	194	7.09	0.35	5.55	7.81

The difference between our result for the Bashi mothers in the Kivu and that of Kon and Mawson for English mothers in Reading and Shoreditch seems to suggest that certain factors (diet?) may have some influence on the lactose content of the milk. Indeed, we used exactly the same method and reagents for the lactose determination. The difference between our work and that of the English authors is one of sampling: they worked on milk samples taken during the morning feed, whereas we worked on 24 hour samples, but no evidence has been found for a diurnal variation in the lactose content of the milk (Morrison, 1952).

TABLE 8 — *Variation of lactose with volume of milk secreted per 24 hour period*

Volume of secretion	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
Between:					
0 ml and 100 ml . . .	4	6.91	0.47	6.10	7.29
100 ml and 200 ml . . .	19	7.01	0.33	6.42	7.50
200 ml and 300 ml . . .	45	7.08	0.37	5.55	7.81
300 ml and 400 ml . . .	42	7.19	0.31	6.08	7.76
400 ml and 500 ml . . .	36	7.15	0.30	6.44	7.55
500 ml and 600 ml . . .	24	7.02	0.34	6.29	7.50
600 ml and 700 ml . . .	13	6.98	0.42	5.84	7.51
700 ml and 800 ml . . .	7	7.03	0.23	6.76	7.48
800 ml and 900 ml . . .	1	6.70	—	—	—
900 ml and 1,000 ml . . .	1	7.41	—	—	—
1,100 ml and 1,200 ml . . .	1	6.74	—	—	—
Total.	193	7.09	0.35	5.55	7.81

THE TOTAL SOLIDS AND THE «SOLIDS NOT FAT» OF THE MILK

Tables 9 and 10 give the variation of the total solids in the milk (excluding colostrum) with the stage of lactation and with the milk yield per 24 hours.

Although there seems to be a gradual decline of the total solids in the milk in the course of the first three months of lactation, a statistical analysis of our results proved that the differences in the total solids-content between the 1st and the 2nd month, the 2nd and the 3rd month and between the 3rd and the 4th month were not significant.

Our mean value for the total solids content of human milk (11.91 g/100 g) is very similar to that of 12.4 g/100 ml given by Macy *et al.* (1953) as a good representative value for most literature reports prior to 1953. If we take the average density of the milk to be 1.031 (Macy, 1953), then our value becomes 12.3 g per 100 ml, which is very close to the 12.4 g/100 ml calculated by Macy from the literature.

TABLE 9 — *Variation of total solids with stage of lactation*

Month	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
1st	19	12.26	1.03	9.94	14.62
2nd	27	12.17	1.14	9.70	14.24
3rd	25	11.92	0.89	10.08	13.63
4th	22	11.71	0.99	9.99	14.10
5th	16	12.14	1.31	9.91	13.89
6th	19	11.83	1.03	9.80	13.45
7th	13	11.64	1.05	9.74	13.29
8th	12	11.77	0.82	10.33	13.46
9th	9	11.64	0.99	9.59	13.16
10th	8	11.16	1.01	9.04	12.58
11th	1	11.23	—	—	—
12th	5	12.46	1.16	10.77	14.41
13th	5	10.61	0.75	9.42	11.78
14th	5	12.38	0.83	11.46	13.85
15th	8	12.24	0.53	11.59	13.25
16th	2	11.63	—	10.42	12.83
17th	5	11.62	0.60	10.84	12.18
18th	1	11.26	—	—	—
19th	1	12.05	—	—	—
20th	1	11.29	—	—	—
22nd	4	12.06	0.54	11.39	12.74
31st	1	14.22	—	—	—
32nd	1	13.10	—	—	—
Total	210	11.91	—	9.04	14.62

So as to make comparisons possible with the work of Kon and Mawson (1950), we have given the «solids not fat» in function of the stage of lactation and in function of the milk yield in Tables 11 and 12.

The «solids not fat» are obtained by subtracting the fat content of the milk from its total solids content. The English authors found an average of 8.93 g per 100 ml of milk for 1,045 samples from Reading and 9.08 g/100 ml for 492 samples from Shoreditch (average for the 2 towns 8.98 g/100 ml) as against our mean value of 8.71 g/100 g, corresponding to 8.98 g/100 ml. These values are iden-

TABLE 10 — *Variation of total solids with volume of milk secreted per 24 hour period*

Volume of secretion	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
Between:					
0 ml and 100 ml. . . .	1	12.24	—	—	—
100 ml and 200 ml. . . .	20	11.53	1.142	9.04	14.41
200 ml and 300 ml. . . .	52	11.95	1.054	9.42	14.24
300 ml and 400 ml. . . .	45	11.90	0.913	9.74	14.62
400 ml and 500 ml. . . .	42	12.03	1.139	9.70	13.89
500 ml and 600 ml. . . .	25	11.83	1.223	9.80	14.10
600 ml and 700 ml. . . .	13	12.05	0.803	10.84	13.33
700 ml and 800 ml. . . .	7	11.99	0.641	11.16	12.78
800 ml and 900 ml. . . .	1	11.47	—	—	—
900 ml and 1,000 ml. . . .	1	12.18	—	—	—
1,100 ml and 1,200 ml. . . .	1	11.75	—	—	—
Total	208	11.91	—	9.04	14.62

tical. They also found a slow fall of the «solids not fat» content of the human milk throughout lactation. The same trend is noticeable in our results, although it is not so clearly marked.

There does not seem to be any variation in the «solids not fat» content of the human milk with the total milk yield per 24 hours.

THE CALORIC VALUE OF THE MILK

The caloric value of the milk was obtained by using the Atwater factors for heat of combustion of dairy products and sugar as quoted by Macy (1953) from Maynard (1944). The number of grams of fat per 100 g of milk were multiplied by 9.25, the lactose by 3.95 and the protein by 5.65.

The variations of the caloric content with the stage of lactation and with the milk yield per 24 hours are given in Tables 13 and 14.

TABLE 11 — *Variation of «solids not fat» with stage of lactation*

Month	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
1st	18	9.00	0.308	8.50	9.76
2nd	26	8.89	0.397	8.35	10.14
3rd	25	8.61	0.388	7.28	9.22
4th	22	8.73	0.531	8.07	10.78
5th	16	8.68	0.303	7.85	9.80
6th	19	8.67	0.226	8.23	9.24
7th	13	8.66	0.273	8.31	9.06
8th	12	8.62	0.252	8.14	8.96
9th	9	8.61	0.245	8.06	8.97
10th	8	8.26	1.020	5.64	9.03
11th	1	8.59	—	—	—
12th	4	8.62	0.698	7.50	9.34
13th	5	8.69	0.550	8.08	9.65
14th	5	8.62	0.330	8.28	9.24
15th	8	8.85	0.432	8.30	9.90
16th	2	8.56	0.120	8.44	8.67
17th	5	8.49	0.145	8.31	8.75
18th	1	8.53	—	—	—
19th	1	8.33	—	—	—
20th	1	4.11	—	—	—
22nd	4	9.54	1.180	8.79	11.57
31st	1	8.56	—	—	—
32nd	1	8.51	—	—	—
Total	207	8.71	—	5.64	11.57

There does not appear to be any systematic variation in the caloric value of the milk either with the stage of lactation or with the volume of milk secreted per 24 hours.

We found an average of 65.7 calories per 100 ml of milk (excluding colostrum). This value is lower than the 71 calories/100 ml of milk calculated by Macy (1953) from the literature, using the same Atwater factors for heat of combustion. The difference is due to : 1) the lower fat content of the milk in this region: we found an average of 3.32 g of fat/100 ml of milk against the 3.8 g/100 ml given by Macy; 2) the use of different factors to convert total nitrogen to

TABLE 12 — *Variation of «solids not fat» with volume of milk secreted per 24 hour period*

Volume of secretion	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
Between:					
0 ml and 100 ml . . .	1	11.57	—	—	—
100 ml and 200 ml . . .	20	8.57	0.732	5.64	9.30
200 ml and 300 ml . . .	52	8.67	0.203	7.50	10.14
300 ml and 400 ml . . .	45	8.76	0.344	8.06	9.76
400 ml and 500 ml . . .	39	8.75	0.500	7.28	10.78
500 ml and 600 ml . . .	25	8.73	0.373	7.98	9.80
600 ml and 700 ml . . .	13	8.69	0.293	8.28	9.02
700 ml and 800 ml . . .	7	8.54	0.291	8.14	9.10
800 ml and 900 ml . . .	1	8.69	—	—	—
900 ml and 1,000 ml . . .	1	8.75	—	—	—
1,100 ml and 1,200 ml . . .	1	8.48	—	—	—
Total	205	8.71	—	5.64	10.78

protein: we used 5.12 and Macy used 6.25. When recalculating Macy's data using the factor 5.12 to make his results comparable to ours, we found an energy value of 69.4 calories for Macy's data.

THE ASH CONTENT OF THE MILK

Tables 15 and 16 give the variations in the ash content of the milk with the stage of lactation and with the milk yield per 24 hours.

There appears to be a steady decline in the ash content of the milk throughout lactation. This is in agreement with the findings of most other workers in this field, as reported by Morrison (1952). Our mean value of 176 mg of ash per 100 g of milk is lower than the average of 0.21 g/100 ml of milk reported by Macy (1953) and is also lower than most of the figures given by Morrison (1952). A detailed analysis of some of the ash constituents is in progress in our laboratories.

Table 13 — *Variation of calories per 100 g of milk with stage of lactation*

Month	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
1st	15	67.4	7.71	53.5	79.6
2nd	22	65.2	8.84	45.2	79.7
3rd	25	62.9	8.12	44.9	79.6
4th	18	61.6	7.68	48.1	84.1
5th	17	64.7	8.27	49.0	79.3
6th	16	64.8	7.48	49.0	77.2
7th	13	62.4	9.45	42.2	77.7
8th	9	63.1	5.49	55.7	75.5
9th	9	60.0	9.02	41.5	74.6
10th	8	62.4	7.17	49.2	74.3
11th	1	58.3	—	—	—
12th	5	67.2	12.74	51.1	86.5
13th	5	52.6	6.08	44.7	62.7
14th	6	68.5	5.60	60.6	78.3
15th	8	65.0	7.12	51.6	75.1
16th	2	62.2	—	51.6	72.7
17th	4	60.1	5.56	54.0	65.7
18th	1	56.5	—	—	—
19th	1	68.6	—	—	—
20th	1	60.4	—	—	—
22nd	3	61.4	4.11	57.2	67.0
31st	1	86.8	—	—	—
32nd	1	75.4	—	—	—
Total. . .	191	63.75	—	41.5	86.8

There does not appear to be a systematic variation of the ash content of the milk in function of the daily milk yield.

THE BETA-CAROTENE AND VITAMIN A CONTENT OF THE MILK

Table 17 gives the vitamin A and the beta-carotene content we found for the milk (excluding colostrum).

We did not find any systematic variation of the beta-carotene and the vitamin A content of the milk with the stage of lactation nor with the total milk yield per 24 hours.

TABLE 14 — *Variation of number of calories per 100 g of milk with yield per 24 hours*

Volume of secretion	Number of samples	Means calories/100 g	Standard deviation	Range	
				Lowest	Highest
Between:					
0 ml and 100 ml . . .	3	65.4	6.45	59.2	74.3
100 ml and 200 ml . . .	19	62.1	9.04	44.9	77.7
200 ml and 300 ml . . .	45	64.6	8.76	41.5	86.8
300 ml and 400 ml . . .	42	63.3	7.89	42.2	79.6
400 ml and 500 ml . . .	35	64.9	8.87	48.1	81.1
500 ml and 600 ml . . .	24	62.1	10.69	45.2	84.1
600 ml and 700 ml . . .	13	64.8	7.64	54.0	78.3
700 ml and 800 ml . . .	7	64.9	5.07	58.0	74.7
800 ml and 900 ml . . .	1	56.7	—	—	—
900 ml and 1,000 ml . . .	1	65.5	—	—	—
1,100 ml and 1,200 ml . . .	1	61.5	—	—	—
Total.	191	63.75	—	41.5	86.8

TABLE 15 — *Variation of ash with volume of milk secreted per 24 hour period*

Volume of secretion	Number of samples	Means grams/100 g	Standard deviation	Range	
				Lowest	Highest
Between:					
0 ml and 100 ml . . .	1	0.151	—	—	—
100 ml and 200 ml . . .	20	0.166	0.035	0.108	0.227
200 ml and 300 ml . . .	52	0.178	0.037	0.112	0.289
300 ml and 400 ml . . .	45	0.179	0.032	0.113	0.268
400 ml and 500 ml . . .	39	0.177	0.029	0.113	0.242
500 ml and 600 ml . . .	25	0.174	0.039	0.106	0.265
600 ml and 700 ml . . .	13	0.174	0.031	0.138	0.247
700 ml and 800 ml . . .	7	0.185	0.028	0.153	0.240
800 ml and 900 ml . . .	1	0.179	—	—	—
900 ml and 1,000 ml . . .	1	0.110	—	—	—
1,100 ml and 1,200 ml . . .	1	0.179	—	—	—
Total.	205	0.176	—	0.106	0.289

TABLE 16 — *Variation of ash with stage of lactation*

Month	Number of samples	Means grams/100 g	Standar deviation	Range	
				Lowest	Highest
1st	19	0.220	0.038	0.110	0.289
2nd.	26	0.200	0.029	0.147	0.268
3rd	25	0.183	0.024	0.130	0.242
4th	21	0.170	0.026	0.113	0.227
5th	16	0 182	0.029	0.113	0.225
6th	18	0.174	0.021	0.141	0 227
7th	12	0.170	0.024	0.124	0.200
8th	12	0 166	0.023	0.137	0.220
9th	9	0.167	0.025	0.129	0.190
10th	8	0.164	0.014	0.140	0.183
11th	1	0.188	—	—	—
12th	5	0 143	0.017	0.115	0.166
13th	5	0 155	0.035	0.127	0.211
14th	6	0.147	0.025	0.113	0.192
15th	8	0.146	0.021	0.108	0.180
16th	2	0.142	0.036	0.106	0.178
17th	5	0.128	0.015	0.110	0.146
18th	1	0.130	—	—	—
19th	1	0.157	—	—	—
20th	1	0.156	—	—	—
22nd.	4	0.143	0.016	0.120	0.162
31st	1	0.199	—	—	—
32nd.	1	0.190	—	—	—
Total. . .	207	0.176	—	0.106	0.289

TABLE 17 — *Beta-carotene and vitamin A content of human milk*

	Number of samples	Means grams/100 g of milk	Standard deviation	Range	
				Lowest	Highest
Beta-carotene	101	21.5	14.9	5.1	89.8
Vitamin A	102	82.4	38.9	18.7	218.4

Macy (1953) reports 27 μg of carotenoids per 100 ml mature milk. We did a chromatographic separation of the beta-carotene from the other carotenoids, whereas Macy's figure gives total carotenoids.

Our vitamin A figure (82.4 μg/100 g of milk) is considerably higher than Macy's average of 53 μg per 100 ml. We have no explanation to offer for this discrepancy. Differences in the methods of determination and in the vitamin A standards used for calibration may contribute to it. The diet of the Bashi mothers is generally fairly poor in foods of animal origin, but they are regular consumers of relatively small quantities of carotene rich palm oil.

We found a positive correlation between the vitamin A and the beta-carotene content of the milk from the mothers in this region ($n = 103$, $r = 0.4357$, $P < .001$).

There is a positive correlation ($n = 101$, $r = 0.3456$, $P < .001$) between the beta-carotene and the fat content of the milk here, and also between the vitamin A and the fat content of the milk ($n = 101$, $r = 0.4116$, $P < .001$).

Table 18 gives the vitamin A and the beta-carotene content of human milk fat.

TABLE 18 — *Beta-carotene and vitamin A content of human milk fat*

	Number of samples	Means micrograms/ gram of fat	Standard deviation	Range	
				Lowest	Highest
Beta-carotene	98	6.6	3.6	1.4	24.4
Vitamin A	97	24.5	10.4	4.3	65.4

There was no systematic variation noticeable in the beta-carotene and the vitamin A content of the human milk fat either with the stage of lactation or with the milk yield per 24 hours. All the results are marked by a very wide dispersion of the figures around the mean.

Kon and Mawson (1950) reported an average vitamin A content of 10.6 μg per gram of milk fat and a mean carotenoid content of 3.63 μg per gram of fat for about 600 milk samples from women in Reading (Engand). The mean fat content of their milk samples was considerably higher (4.66 g/100 ml) than ours (3.32 g/100 ml), which was probably partly due to their sampling technique.

We found a positive correlation between the vitamin A and the beta-carotene content of the human milk fat ($n = 97$, $r = 0.2705$, $P < .01$).

DISCUSSION AND CONCLUSIONS

Table 19 gives a comparison of the values for the major components of human milk excluding colostrum as gathered from most of the literature by Macy (1953), as determined by Kon and Mawson (1950) in the South of England and as found by us among the Bashi in the Kivu.

TABLE 19 — *Comparison of the major components of human milk as determined in the Kivu and elsewhere. All figures are given as grams/100 ml of milk*

	Total nitrogen	Fats	Lactose	Total solids	Solids not fat	Calories	Ash
Macy *et al.* (1953) . . . Literature average	0.227	3.8	7.0	12.4	8.6	69.4 (*b*)	0.21
Kon + Mawson (1950). . Sout of England	0.193	4.66 (*a*)	6.93	13.6	8.98	76.1 (*c*)	—
Roels + Trout. . . . . Kivu, Belgian Congo	0.184	3.32	7.31	12.3	8.98	65.7	0.18

(*a*) Determined on samples of milk taken in the course of the morning feed.
(*b*) Recalculated by us, using the factor 5.12 to convert total nitrogen to protein.
(*c*) High because of the high fat content.

We found the average caloric value, the nitrogen, fat and ash content to be lower and the lactose content to be higher in the Kivu than the corresponding mean values reported by Kon and Mawson for England, and by Macy mainly for European and American mothers. It is remarkable that both the fat and the protein should be lower here than that reported (mainly) from regions with higher dietary standards. This observation led us to examine the relationship between the fat and the protein content of the milk from the mothers here.

We found a very strong positive correlation between the «protein» (= total nitrogen × 5.12) content and the fat content of the milk ($n = 224$, $r = 0.4426$, $P < .001$) from the Bashi mothers in the Kivu. This means that for this region a milk sample with a low fat content tends to have also a low «protein» content, that the fat content of the milk and the «protein» content of the milk rise together, and that a milk sample with a high fat content tends to have at the same time a high «protein» content. As far as we know, this correlation has not been observed before in human milk and we think that it would be important if some of the workers in this field would examine their data in this light, to see whether the same correlation is observed elsewhere, with mothers having a completely different diet. It cannot be calculated from the literature on the subject because only mean values for the various components are published and not the reports of analyses of individual samples. This observation may help towards a better understanding of the relationship between the diet of the mothers and the nutritive value of their milk, and perhaps even of the mechanism of the milk secretion in the mammary gland. Breirem (1957), in his literature survey of the relation between the nutritional value of cow's milk and the nutrition of the animal, reports that Møllgaard and Lund, Steensberg, Kay, Breirem and Saarinen have observed that deficiency of energy and/or protein in the diet of the cow will reduce *fat and protein* content of the milk. Janz *et al.* (1957) report a similar observation made by Escudero and Pierangeli for human milk: these authors reported that mothers with a diet rich in proteins secreted milk a *high protein and fat content.*

These reports bring indirect circumstancial evidence for the existence of this positive correlation between the fat and protein content of milk which we have observed so clearly in our work.

We have not observed in this work whether the fat and protein content of the mature milk from one mother remains fairly constant throughout lactation, as nearly all the mothers whose milk we have examined came only once to give their milk for this project.

This should be a very interesting subject for further research. If it were true, which seems likely in view of the observations reported by Breirem (1957) and Janz (1957), which we have already quoted, then its implications for the nutritional benefits a baby will derive from his mother's milk are quite important. It would mean that some babies would tend to get regularly milk which is low in protein *and* in fat. As their mother's milk is the only high quality food babies generally get, the repercussions on their growth and health may be considerable.

We found no evidence of any correlation between the «fat plus protein» content of the milk and the lactose content of the milk ($r = 0.176$). As we found no indication of any systematic change of either the fat or the protein content of the milk with the yield per 24 hours, it does not look as though there would be a systematic compensation for low «protein and fat» content of the milk through higher secretion. Calculation of the correlation coefficient ($r = 0.0305$) proved that there was no evidence for any relation between «fat plus protein» content of the milk and daily milk yield.

Here again, further research seems indicated to establish whether the same women tend to secrete relatively similar quantities of milk every day, since this may influence the daily nourishment the baby receives from his mother's milk.

SUMMARY

As part of a research program on milk from African mothers undertaken in the Nutrition Laboratory of the «Institut pour la Recherche Scientifique en Afrique Centrale» at Lwiro (Kivu, Belgian Congo) we have determined total fat, total nitrogen, lactose, total solids, ash, vitamin A and carotene in 225 samples of all the milk secreted during a 24 hour period, by Bashi mothers, living in their customary rural environment. The Bashi tribe lives on the Eastern shore of Lake Kivu in the Belgian Congo, between 2° 10′ S and 2° 40′ S, about 29° E of Greenwich, at an altitude varying from 1460 m to 2000 m above sea level. The ages of the mothers ranged from 17 to 40 years. Their staple foods are beans, sweet potatoes and beer made from bananas.

The results have been grouped according to the stage of lactation, from birth until over 2 year's old, and according to the total volume of milk secreted during the 24 hour period. There is a marked decrease in the total mineral (ash) content of the milk throughout lactation. The total nitrogen (and hence the protein content) of the milk decreases sharply until it reaches a steady value at the 3rd to the 4th month after birth. There seems to be little variation in the fat and lactose content of the milk, either with the stage of lactation or with the total volume of milk secreted per day.

We found the following average composition for the mature milk (results in grams per 1000 g of milk):

- — 11.88 g of total solids (excluding 1st month of lactation)
- — 3.21 g of fats (excluding 1st month of laction)
- — 0.17 g of ash (excluding 1st month of lactation)
- — 7.09 g of lactose (excluding 1st month of lactation)
- — 0.167 g of total nitrogen whence (excluding first three months of lactation)
- — 0.855 g of proteins (nitrogen × 5.12) (excluding first three months of lactation)

The mean beta-carotene content of the milk was found to be 21.9 µg per 100 g of milk and the average vitamin A content was 82.4 µg per 100 g of milk.

We found a strong positive correlation between the «protein» and the fat content of the human milk in this region.

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SOME ADVANCES IN THE KNOWLEDGE OF PROTEIN MALNUTRITION

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In spite of the vast amount of work that has been done on protein malnutrition of infants since the end of the war, many problems are still unsolved. In this paper I shall consider three questions that have been occupying us in Jamaica in the last few years. These are:

1 — The determination of the degree of protein depletion
2 — The interpretation of the changes in water and electrolytes
3 — The cause of death

1 — THE DETERMINATION OF THE DEGREE OF PROTEIN DEPLETION

One of the salient, if admittedly obvious, characteristics of protein malnutrition is that the body is depleted of protein. Undoubtedly other factors influence the picture, such as vitamin deficiencies and, perhaps even more important, the level of calorie intake. But it seems to me that protein depletion must underlie the whole spectrum of clinical cases covered by the term protein malnutrition, from classical kwashiorkor at one end of the scale to marasmus at the other [1]. It would be useful to be able to measure the degree of depletion, for two reasons: first, to compare the severity of different cases; secondly, as a background for studying the differences in pattern that may be found between cases of equal severity. Until this has been achieved we are in the position of someone trying to study anaemia without means of measuring either haemoglobin concentration or red cells indices.

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[1] WATERLOW, J. C., CRAVIOTO, J., & STEPHEN, J. M. L. — *Adv. Prot. Chem.*, **15**: 131, 1960.

We have attacked the problem by three methods, but so far with only limited success.

Our first approach was by tissue analysis. We measured the protein and DNA content in specimens of liver and muscle obtained by biopsy in living subjects, and at necropsy in fatal cases. DNA was used as a standard of reference on the assumption that the amount of this substance per cell is not affected by malnutrition — i.e., that the cytoplasm shrinks round the nucleus. The ratio of protein N to DNA-phosphorus is therefore a measure of the amount of cytoplasmic protein in the cell.

Table 1. In the cases that did not die, a biopsy taken about one week after admission to hospital was compared with one taken after one to three months treatment. If the final biopsy figures are taken as «normal», Table 1 shows that in the malnourished state 30 to 40 % of the tissue protein was lost. Similar results were found in the fatal cases.

TABLE 1 — *Nitrogen content in relation to DNA of liver and muscle in malnourished infants*

	N/DNAP (mg/mg)		Percent decrease
	Initial biopsy	Final biopsy	
Liver	49	83	41
Muscle	237	343	31

We had hoped that this type of analysis, which is analogous to the measurement of haemoglobin concentration, might enable us to grade the severity of depletion in the individual case. This expectation was not fulfilled; the tissues, unlike blood, are not homogeneous, and in these small specimens (our liver samples weigh about 5 mg and muscle samples about 100 mg) there is a large sampling error.

Thus, although differences between the means of groups are significant, those between individuals are not. All that the results show us, therefore, is the *order of magnitude* of the protein depletion in the severe case.

Our next approach was an old-fashioned one — the measurement of 24-hour creatinine excretion as an index of total muscle mass. This was suggested by Dr. Arroyave, of INCAP, who found a striking difference in the creatinine excretion of poor rural Guatemalan children and well-nourished controls. In order that different cases may be compared, it is convenient, as Arroyave has suggested, to relate the creatinine output to body height.

Graph 1 shows how the output, expressed in this way, increases during the period of treatment in hospital. These figures are taken from a group of cases of kwashiorkor or marasmic kwashiorkor.

Table 2 shows that the muscle mass, as judged by the creatinine output, more than doubled during the period in hospital. It increased by a factor of 2.3 (from 0.8 mg to 1.8 mg per centimeter — third column of Table 2[1].

TABLE 2 — *Creatinine output by malnourished infants (mg/day)*

Days after admission	Number of cases	Creatinine	
		mg / day / cm	mg / day / kg
10–19.	15	0.80	9.1
20–39.	13	1.04	11.9
40–59.	11	1.50	14.6
60 +.	6	1.83	14.95

The interesting point is that this increase is much greater than the increase in body weight over the same period: on the average, the final weight was 1.6 times the initial weight. In other words, in the malnourished state the deficit in muscle mass is greater than one would suspect from the deficit in body weight. This fits in with the

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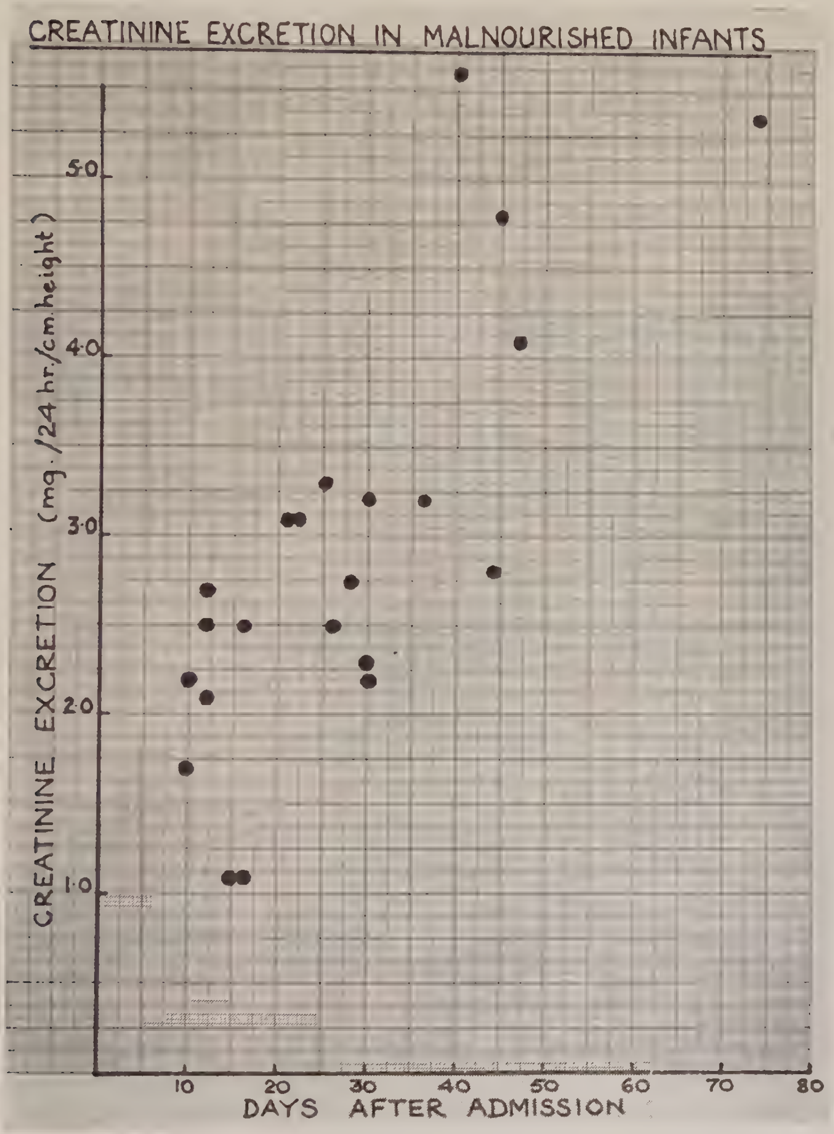
[1] STANDARD, K. L., WILLS, V. G., & WATERLOW, J. C. — *Amer. J. Clin. Nutr.*, 7: 271, 1959.



Graph 1

results of tissue analysis: the increase in the ratio of protein to DNA during treatment was also greater than the increase in body weight. It is easy to see how this comes about: certain tissues, such as brain, skeleton, and connective tissues generally, are not affected by malnutrition, or much less affected than the more labile organs with a higher protein-turnover rate, like liver and muscle. If some organs are depleted less than the average, others must be depleted more. Moreover, although the initial body weights I have referred to were taken after the loss of oedema, it has been shown that even at this stage there is an increase in total body water. The loss of the excess water during recovery masks the increase in tissue mass.

Again, from our work so far it does not seem likely that the 24-hour creatinine output will be a very reliable index of the extent of depletion in the individual case; the excretion seems to be affected by a number of variables, so that not too much reliance can be placed on a single 24-hour specimen.

The third method we have used is the measurement of total body water (TBW) with tritiated water. This work has been done by Dr. Roger Smith, and the results he has obtained so far are shown in Table 3 [1].

TABLE 3 — *Total body water — Percent of body weight at different stages of treatment*

Time	Number of cases	TBW (percent)	Range
In presence of oedema	12	84.3	74.6-91.5
Within 35 days of loss of oedema	15	75.2	54.4-86.0
On recovery	7	62.6	53.9-70.1

As I said above, even after the loss of oedema a considerable increase in body water still persists. For the present purpose the most useful way of looking at these results is to consider, not the percentage of body water, but the *absolute* amount, and by subtracting this from the body weight, to obtain a figure for the total body solids. An unknown proportion of this total will be fat.

Graph 2 shows in a typical case the increase in total solids calculated from the body water. (The height of the whole column represents body weight, the shaded area, body solids, and the clear area, body water; the dotted line indicates the amount of water that would correspond to this amount of solids if the ratio of water to solids were normal.)

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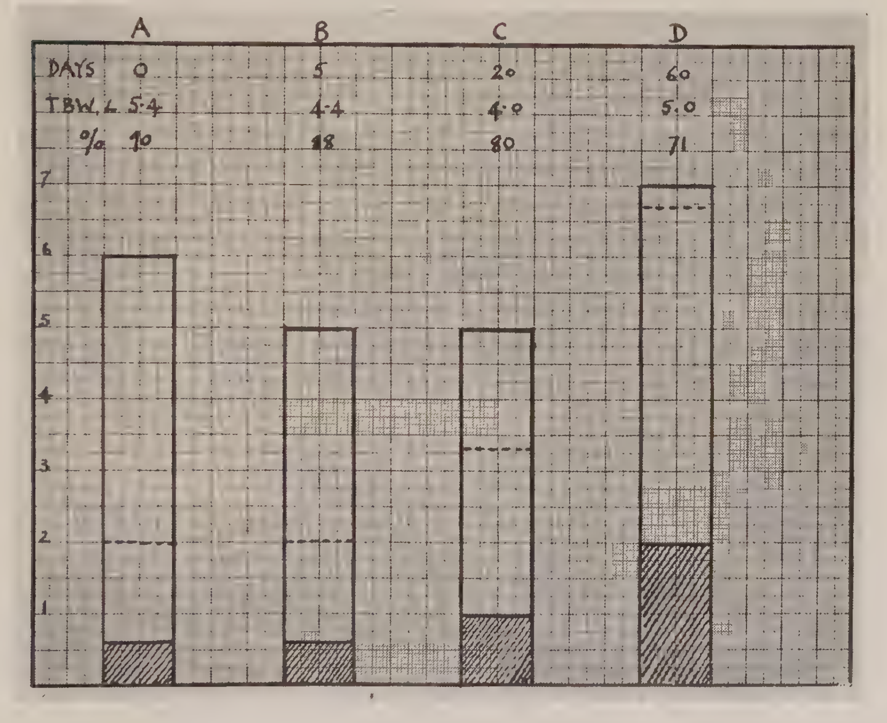
[1] SMITH, R. — *Clin. Sci.*, **19**: 275, 1960.

The point I want to emphasize is the exceedingly severe reduction in total body solids, and *a fortiori* of active tissue mass, in the malnourished state. In this child (Graph 2) at the time of admission the total solids amounted to 0.6 kg, which is only 30 % of the non-fat solids of a normal child of the same age.



Graph 2

These results, obtained by three methods, agree in indicating the seriousness of the tissue loss and protein depletion in these babies as a group. It cannot be said, however, that the methods are yet of much practical value for the diagnosis and treatment of the individual case. Creatinine output and measurement of total body water could, however, without too much difficulty, be used for the study of marginal malnutrition in the field.

2 — THE INTERPRETATION OF THE CHANGES IN WATER AND ELECTROLYTES

The Table 3 and Graph 2 showed that in these malnourished babies there is an increase in total body water even in the absence of clinical oedema. We have not attempted to measure extracellular fluid volume, since there is no simple and accurate method for this; therefore we do not know from these studies on the whole body whether or not some of the excess water is intracellular. However, last year Gomez and his colleagues in Mexico published a series of papers, in which they showed by analysis of muscle that, even in clinically dehydrated cases, there is an increase of intracellular water.

Table 4. The increase in water is accompanied by an increase in total body sodium. This was first shown by Hansen in Cape Town, who observed a large sodium diuresis when oedema is lost. The Mexican workers found an increase in muscle sodium content, and, by calculations based on the assumption that all chloride is extracellular, showed that in some cases there must be an accumulation of sodium inside the cells.

TABLE 4 — *Composition of muscle in malnourished infants (Data of Gomez* et al., *1957)*

	Water (g/kg)		Meq./litre intracellular H_2O	
	Extracellular	Intracellular	K	Na
Control	135	224	159	10
Dehydrated	101	294	116	35
Oedematous	184	266	122	13

In our muscle biopsies, we found a slight increase in sodium, with a normal ratio of *Na* to *Cl*, suggesting that the increase is extracellular. *Table 5*, bottom line. These findings, however, are not conclusive, since our biopsies were done primarily for the measurement of protein content after the acute electrolyte disturbances had subsided.

Although the total body sodium is increased, the sodium *concentration* in serum is usually low, sometimes as low as 110 meq. per litre. From this it follows that both extra- and intra-cellular fluids must be hypotonic, since modern evidence has shown very clearly that the two compartments are isosmotic. Gomez and his co-workers, by direct measurements of the osmotic pressure of serum, have shown that hypotonicity of the body fluids is a constant finding in these cases.

TABLE 5 — *Composition of muscle in malnourished infants*

Biopsy	Initial	Final
Body weight (percent of ideal weight) . . .	53	68.5
Water content (percent)	77.6	75.7
Non-collagen nitrogen (mg/mg DNAP) . .	237	343
«Supernatant» P (µg/mg NCN)	26.3	32.3
K (µeq/mg NCN)	2.43	2.82
Na/Cl (µeq/µeq)	1.42	1.365

In addition to the hypotonic expansion of the body fluids, with sodium accumulation, there seems usually to be a depletion of potassium. This was shown by Hansen by the cumulative balance technique. The potassium concentration tends to be low in both serum and muscle. Recently we have measured the total exchangeable potassium in a small number of cases by means of isotopic potassium. The results showed that in two cases about one-third or more of total body potassium was lost. We do not yet know whether the potassium depletion results simply from diarrhoea and a reduced intake, or whether there is a failure of the renal tubule to conserve potassium.

In many of our cases we have also found evidence of phosphorus depletion. All those who have measured the serum inorganic phosphorus have found it to be low. Muscle phosphorus content also tends to be low (Table 5, 4th line), and balance measurements show that the organism has a tremendous avidity for phosphorus; it retains relatively much more phosphorus than nitrogen compared with the amounts of these two elements in normal muscle, provided that enough phosphorus is obtainable from the food (Table 6). In other words, there seems to be a depletion of intracellular constituents — protein, potassium and phosphorus. We have no information about magnesium.

What cause can we postulate for these disturbances in water and electrolytes? Almost certainly the chain of events is complex. Hypoproteinaemia must be an aggravating factor, but cannot be the sole cause, since it does not explain the hypotonicity. Gopalan and Heller have put forward the attractive idea that there may be an increase in circulating anti-diuretic substance. The reason for this, they suggest, is that the damaged liver is unable to inactivate the hormone. This hypothesis would explain the statistical association that we have found in Jamaica between the presence of oedema and fatty liver. Against the ADH hypothesis is the accumulation of the sodium, which is not a characteristic effect of this hormone. The increase in water and sodium, and the decrease in potassium, could all be produced by excessive aldosterone secretion, such as occurs in certain other forms of oedema. We are at present investigating this possibility. But even if aldosterone secretion is excessive, the question remains, what is the stimulus?

TABLE 6 — *Relation between P/N in milk and P/N retained in the early stage*

P / N in milk (mg / g)	P / N retained (mg / g)
175	167
141	121
100	98
65	49

P / N in muscle : 68 mg / g

Finally, it seems to me possible that the prime cause may lie, not in altered hormone activity, but in altered cell function. The retention of potassium in the cell and the exclusion of sodium depend on a source of energy. In recent studies on the liver we have found evidence of a reduction in the rate of oxidative phosphorylation, which is the main mechanism of energy transfer[1].

3 — THE CAUSE OF DEATH

In our ward, in which admittedly we take mainly the most severe cases, 20 % of the malnourished babies die. What is the cause, or perhaps it would be more accurate to say the mode, of death? Many answers to this question have been put forward, only to be discarded one by one. Infections, of course, are important, but in my opinion are only aggravating factors. The prophylactic use of antibiotics has not, in our hands, made much difference to the mortality. One of the earliest theories was that there was a disturbance of carbohydrate metabolism with hypoglycaemia, but this is not supported by any real evidence. Then, more than 10 years ago, I suggested that death might result from liver failure, because of the huge fatty infiltration of the liver that is characteristic of our cases in the West Indies. Further experience has not supported this idea. A deposition of fat up to 50 % of the wet weight of the liver seems to be quite compatible with life, and the degree of fatty infiltration bears no relation to the general severity or the prognosis.

Next, potassium deficiency has been incriminated. When this is not recognized and treated, it may well be the cause of death. But in our experience cases may still die in spite of potassium supplementation, at a time when the serum potassium level has been normal for several days. In muscle samples taken immediately after death, the potassium content has been normal, or no lower than in cases that survive. Recently we have been impressed by the low serum phosphate levels in the severe cases; a phosphorus concentration of less than 2 mg per 100 ml, compared with the normal of 6 mg to 8 mg, seems to be of grave omen, and we are now treating these cases with extra phosphorus as well as potassium. Whether this will have any effect on the mortality remains to be seen.

Finally, it may well be that the depletion of cellular constituents has led to an irreversible failure. If, as we have shown, the cell has lost 40 or 50 % of its protein, this loss must include enzymes. The machinery may therefore be so badly damaged that recovery is impossible. I have already mentioned a fall in the rate of oxidative phosphorylation in the liver, which seems to be caused by some kind of damage to the mitochondria. For safety's sake we do not do liver biopsies when the child is in a very bad state; usually the biopsy is postponed for 7 to 10 days until the general condition is a little better. If even under these conditions there is a reduction in phosphorylation, it would be reasonable to suppose that in the very severely ill child the reduction would be greater. Clearly if the rate of oxidative phosphorylation falls below the minimal level necessary for maintaining the integrity of the cell, the result must be an irreversible catastrophe, i.e., death.

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[1] WATERLOW, J. C. — *Proc. Roy. Soc. B.*, **155**: 96, 1961.

One of the conditions of recovery is that the body must be able to synthesize new protein. If there is a failure of the enzymatic machinery of the cell, one would expect that this function would be destroyed, and the organism would be unable to retain or utilize nitrogen. We have tried to investigate this point by the balance method. Some cases, even in the day or two before death, do seem to retain reasonable amounts of nitrogen. However, conditions make accurate results difficult to achieve; the baby usually has diarrhoea, is vomiting, and refuses food. Moreover, in the presence of an increased body water, appreciable amounts of nitrogen might be retained as free amino-acids or urea without being incorporated into protein. We are trying to get round this difficulty by giving radio-active methionine parenterally. Then vomit and stools can be ignored, and all that is necessary is to collect the urine. So far only one fatal case has been investigated in this way; it died 30 hours after the injection; at the time of death portions of plasma, liver and muscle all showed a good incorporation of the labelled amino-acid. If this case is typical, yet another theory about the cause of death — an irreversible failure of protein synthesis — will have to be jettisoned.

In this paper I have had time to deal with only a few of the problems raised by the syndrome of severe protein malnutrition. Much has had to be left out. I recognize also that these severe cases are only a small proportion of the whole. From the point of view of public health and the nutrition of a population, the most urgent objective must be to find means of recognizing and assessing the mild and marginal cases.

ANAEMIA IN KWASHIORKOR

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Kwashiorkor is a disease of infancy and childhood found in all the backward countries where ignorance is rife, poverty universal and first class protein expensive and in short supply. In South Africa it is confined to the coloured races In Durban the clinical picture as recorded elsewhere (Walt *et al.*, 1950) is very similar to that in other countries. The principal features are generalised oedema, dermatoses, dyspigmentation of hair and skin, hypoproteinaemia and retardation of physical development. As occurs elsewhere, it is very commonly complicated by such infections as dysentery, gastro-enteritis, pneumonia, upper respiratory infections and tuberculosis; although helminthiasis is common, it seldom causes serious ill health or anaemia.

Anaemia was found in almost every child with kwashiorkor studied by us and it is the anaemia in our patients which has interested us considerably. Anaemia is diagnosed when the haemoglobin is below 11 g per 100 ml because this is the level one would like one's patients to achieve and is also the level suggested by Trowell (1956).

Various types of anaemia have been described by many workers in a syndrome aparently identical with kwashiorkor. Some anaemias are normochromic and normocytic (Altmann and Murray, 1948; Adams, 1954), others megaloblastic (Amato, 1946; Gerbasi and Burgio, 1955), and others hypochromic (Trowell and Simpkiss, 1957). Almost every worker has attributed the anaemia to different causes such as associated infections or lack of protein (Woodruff, 1955), or iron or folic acid. In previous studies (Walt *et al.*, 1956 and 1957) it became apparent that patients almost invariably remained anaemic even after recovery from kwashiorkor and despite active treatment

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directed at the anaemia. It is this persistent anaemia that I wish to discuss with particular reference to the common features it presents during therapy. It is hoped that these findings may indicate lines for further investigation. I propose to discuss briefly the megaloblastic anaemia which also occurs in our cases of kwashiorkor.

TYPES OF ANAEMIA IN KWASHIORKOR

The two principal types of anaemia are:

1. Megaloblastic anaemia.
2. Normochromic normocytic anaemia.

1. MEGALOBLASTIC ANAEMIA. — Megaloblastosis is a common complication of kwashiorkor and it is estimated to occur in 15 % to 20 % of our patients. In 1957 we reported megaloblastosis in 9 % of all patients admitted to our wards over a period of 3 months (Walt *et al.*, 1957). Any studies of this disease should take into account the seasonal variations in incidence because we have shown both kwashiorkor and megaloblastosis to be more common in the summer months (Walt *et al.*, 1956). Thus, in a series of 35 patients with kwashiorkor presently under investigation, 25 were seen during the summer months (November 1957 to March 1958) and of these 6 (25 %) had megaloblastosis on admission. On the other hand, megaloblastosis has not been found in any of the 10 patients admitted during the autumn and winter (April, May and June 1958). When megaloblastosis is discovered on routine bone marrow examination in mildly anaemic patients, it is not usually the basis for the anaemia because treatment with folic acid causes disappearance of the megaloblasts but not of the anaemia. However, when a patient with kwashiorkor is admitted in a severely anaemic state, it is almost invariably due to a megaloblastic dyshaemopoiesis which often tends to occur suddenly. Such patients apear to be very ill, are exceptionally pale and usually have haemoglobin levels varying from 2,5 g to 5 g per 100 ml. This «crisis», which is characterised by a drop of 3 g to 4 g of Hb. per 100 ml in 24 hours may occur in any patient with megaloblastosis. The bone marrow is then almost entirely mega-

loblastic, but only occasional megaloblasts may be found in the peripheral blood. Although folic acid therapy will cure the megaloblastosis within 96 hours (Walt *et al.*, 1956), blood transfusion is thought to be necessary to save life.

2. NORMOCHROMIC NORMOCYTIC ANAEMIA — This is the anaemia common to all our patients with kwashiorkor, including those who have recovered from megaloblastosis; it is mild the haemoglobin level varying from 7 g to 10 g per 100 ml. Despite adequate treatment with vitamins and high protein diets and apparent recovery from kwashiorkor, the haemoglobin level is very slow to rise. It is this slow haematological response to treatment that I have been able to observe in 23 cases from November 1957 to June 1958 that I now wish to discuss (Table).

RESPONSE OF ANAEMIA TO THERAPY

All cases with tuberculosis, dysentery and in need of blood transfusions were eliminated from the series.

TREATMENT.

Routine. — For the first 14 days each patient is given as much lactified skimmed cow's milk as he will take, our nurses being trained to encourage and coax the patient in a kindly and patient manner. Thereafter, a full ward diet is offered. Potassium chloride 5 g three times daily, procain penicillin 300,000 units by intramuscular injection and a triple sulphonamide 90 mg per lb. body weight daily is given for the first 7 days. Folic acid 7.5 mg intramuscularly daily is given routinely for 3 days after admission and thereafter 5 mg three times a day by mouth. Vitamin B complex is given orally from the fourteenth day and vitamin C 50 mg daily throughout the hospital stay.

Special therapy in this series. — Alternate patients were also given a protein supplement of «Tervamix» (group A), a powder containing 68 % milk protein obtained from skimmed milk, which supplied an extra 1 g protein per lb. body weight daily. Daily observations were made for a period of 28 days (Table).

Summary of observations on 23 cases of kwashiorkor

Case number	Patient	Race	Admission date	Age in months	Sexe	Admission haemoglobin (grams per 100 mc.)	28th day haemoglobin (grams per 100 mc.)	Bone marrow	Serum proteins						Associated infections	Hospital day of infection	«Tervamix» given
									Admission			Discharge					
									Total	Alb.	Glob.	Total	Alb.	Glob.			
9 . .	B. P.	Af.	23–11–57	18	F	12.0	7.2	N	6.3	2.8	3.5	6.6	2.9	3.7	Gast.-ent.	1	
															U. R. I.	16	No
															Furunculosis	24	
10 . .	A. M.	Af.	24–11–57	12	F	8.5	7.0	N × 2	4.4	1.7	2.7	7.2	2.9	4.3	U. R. I.	15	
															Pneumonia	18	Yes
11 . .	T. M.	Af.	29–11–57	25	F	11.3	9.2	N	4.6	1.8	2.8	7.1	3.0	4.1	U. R. I.	12	No
13 . .	S. M.	Af.	2–12–57	16	M	9.5	6.6	N	5.0	1.4	3.6	6.8	3.0	3.8	Gast.-ent.	1	
															U. R. I	8	No
															Stomatitis	11	
15 . .	Z. M.	Af.	9–1–57	24	M	10.5	10.1	N	5.8	1.8	4.0	7.2	3.2	4.0	U. R. I.	4	No
															Pneumonia	21	
16 . .	M. T.	I.	12–1–58	15	F	12.8	11.1	N	4.3	1.0	3.3	7.6	3.7	3.9	Nil	—	Yes
19 . .	O. D.	Af.	21–1–58	19	F	9.7	8.4	Mild M	3.3	1.0	2.3	7.1	2.7	4.4	Gast.-ent.	3	No
															U. R. I.	9	
20 . .	T. M.	Af.	21–1–58	36	F	11.8	9.0	N	4.4	1.3	3.1	7.8	3.0	4.8	U. R. I.	11	Yes
22 . .	B. M.	Af.	13–2–58	25	M	10.2	8.8	N	3.6	0.9	2.7	6.4	3.1	3.3	Nil	—	Yes
23 . .	T. G.	Af.	22–2–58	60	F	8.9	7.5	Mild M	4.8	1.1	3.7	7	2.5	4.5	U. R. I.	10	No
															Pneumonia	19	
24 . .	A. D.	Af.	24–2–58	21	F	10.7	10.0	Mild M	3.9	1	2.9	6.4	2.6	3.8	Nil	—	Yes
25 . .	N. N.	Af.	24–2–58	16	F	10.3	9.3	N	3.0	0.7	2.3	6.0	2.4	3.6	Nil	—	No
26 . .	D. S.	Af.	26–2–58	20	F	7.5	10.2	N	2.8	0.6	2.2	6.0	2.6	3.4	Pneumonia	1	Yes
28 . .	E. C.	Af.	7–3–58	22	M	8.6	7.8	N	4.0	1.5	3.5	6.5	2.4	4.1	U. R. I.	1	Yes
32 . .	S. K.	Af.	26–3–58	24	M	12.0	8.2	N	4.1	1.0	3.1	6.7	2.1	4.6	U. R. I.	1	Yes
															Stomatitis	5	
33 . .	S. N.	Af.	28–3–58	15	M	12.0	7.6	N	4.2	1.1	3.1	6.2	2.5	3.7	U. R. I.	15	No
34 . .	S. M.	Af.	17–4–58	24	M	9.5	11.0	N	4.0	1.1	2.9	6.2	2.2	4.0	Nil	—	Yes
38 . .	B. H.	Af.	5–5–58	17	F	9.0	10.5	N	4.0	1.2	2.8	6.2	2.6	3.6	U. R. I.	1	Yes
39 . .	Z. C.	Af.	6–5–58	36	F	12.0	11.6	N	4.1	1.3	2.8	5.5	2.3	3.2	Nil	—	No
40 . .	N. N.	Af.	7–5–58	6	M	11.2	10.0	N	3.4	1.1	2.3	6.2	2.5	3.7	U. R. I.	1	Yes
41 . .	J. M.	Af.	8–5–58	30	M	10.1	9.8	N	4.2	1.5	2.7	7.0	2.4	4.6	U. R. I.	11	No
43 . .	F. M.	Af.	28–5–58	36	F	10.0	11.2	N	5.0	1.6	3.4	7.2	2.7	4.5	Nil	—	No
44 . .	P. M.	Af.	29–5–58	18	M	8.4	11.4	N	3.2	1.1	2.1	6.5	2.4	4.1	Stomatitis	5	Yes

Af. = African.
I. = Indian.

N = Normoblastic bone marrow.
M = Megaloblastic bone marrow.

U. R. I. = Upper respiratory infection.
Gast.-ent. = Gastro-enteritis.

RESULTS.

Up to the time of writing 36 patients have been admitted and of these 9 (25 %) have died. Of the 27 survivors 4 required blood transfusions and were therefore excluded, leaving 23 cases for analysis. Fig. 1 shows how the initial haemoglobin level, estimated on admission, was misleadingly high and how it fluctuated over 28 days. The mean haemoglobin on admission was 10.3 g percent. Despite resolution of clinical signs within 28 days of initiating therapy the mean haemoglobin actually dropped being only 9.2 g percent, revealing a mean difference of 1.1 g per 100 ml. In 18 patients the haemoglobin was still below 11 g percent 28 days after commencement of treatment; 9 of these had received the standard high protein diet only while 9 had had an additional protein supplement as well. Of the 5 patients with a haemoglobin of 11 g percent or more 3 had been supplemented with protein.

Serum proteins. — In all cases the serum protein rose rapidly during the first two weeks of treatment from a mean of 4.1 g percent to 4.8 g percent after one week and then to 5.9 g percent by the fourteenth day (Fig. 1). There was no significant difference between

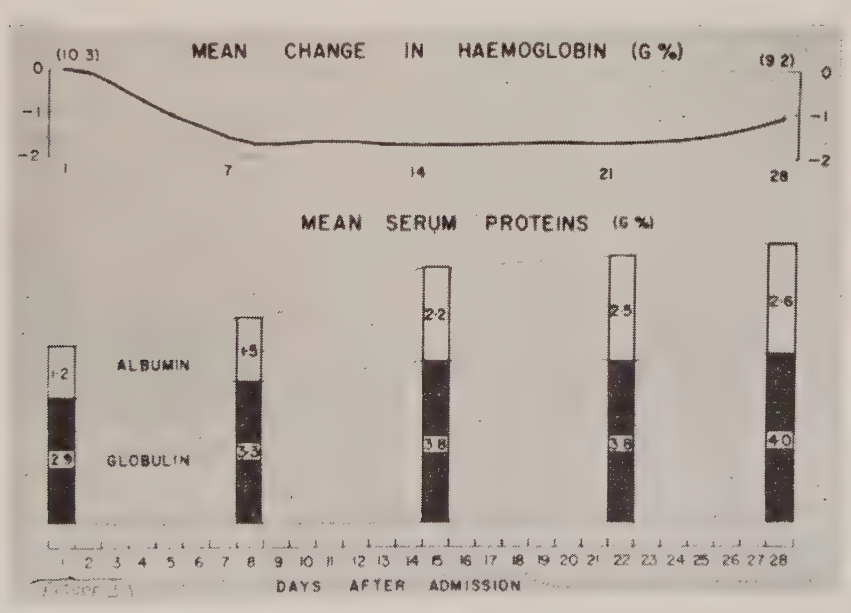

Fig. 1

the mean serum protein levels in those cases who received supplements of protein (group A) and those who did not (group B).

DISCUSSION

The results reported here once more draw attention to the presence of a mild anaemia in kwashiorkor, which is very slow to respond to adequate protein-rich diets fortified with folic acid. Profound metabolic alterations undoubtedly take place during the development of the disease as well as during the period of recovery. These metabolic processes are likely to be dependent oné upon the other and although sufficient protein may be available other substances are probably also necessary for the rebuilding of tissues, the production of enzymes, the absorption and utilisation of vitamins and the mobilisation of iron, folic acid, etc.

MEGALOBLASTOSIS.

Megaloblastosis can be eliminated from the bone marrow very rapidly by administering folic acid. It is assumed therefore to be due to a specific deficiency through lack in the diet of such folic acid containing foods as green leafy vegetables, cauliflower, kidney and liver. Moreover, what little folic acid is ingested is probably poorly absorbed due to associated diarrhoea. Vomiting, a common symptom of kwashiorkor on admission to hospital, may also prevent adequate intake. I have seen a patient, in whom megaloblastosis was resolved by intramuscular folic acid, relapse three weeks later because of severe chronic constipation in spite of taking folic acid regularly by mouth.

Various possibilities have been considered to explain the megaloblastic «crisis» that may occur. A possible explanation is the shortening of the survival time of the red blood cells due to faulty red cell construction resulting from either a lack of haemopoietic substances or from some toxic effect upon the bone marrow resulting from infection. If so, it is possible that many red cells die suddenly, thus causing severe anaemia in a manner comparable to that described by Emery and Lemmon (1954) for the crises in congenital spherocytic anaemia.

The haemoglobin pattern. — Haemoglobin estimation is a simple and useful method of measuring anaemia, but, in kwashiorkor, the initial reading is misleadingly high because of haemoconcentration. Metcoff *et al.* (1945) showed, in severely protein deficient rats, that there was an associated haemoconcentration and a decrease in total circulating haemoglobin because of a reduction in total blood volume. We have also found marked changes in haemoglobin concentration occurring so rapidly that a change in blood volume can be the only satisfactory explanation. Once the patient has had a diuresis and cure has been initiated, the haemoglobin level remains fairly constant (Fig. 1).

Anaemia of infection. — Kwashiorkor predisposes to infection, which very commonly is the reason for the patient's admission to hospital. Our cases therefore are all ill and liable to spread or contract infections in the wards. About 75 % have an acute infection at some time during their illness. Many fail to respond to antibiotic therapy. Virus infections occur frequently, measles being an especially dangerous disease. Those with uncomplicated kwashiorkor respond quickly to treatment and, although the haemoglobin follows the expected pattern of a fall and a gradual rise thereafter, the ultimate rise is more rapid than usual and the haemoglobin may reach 11 g percent by the end of the fourth week of treatment. Many workers have stressed the significance of malaria, hookworm and helminthiasis in their patients, but these infections are not significant in our cases. However, I believe that in Durban gastro-enteritis, dysentery and other acute infections are of greater importance. Adams (1954) did not report the incidence of infections in his cases, while of the 9 young children studied by Woodruff (1955), 8 had ascites — a very rare finding amongst our cases. Josephs (1936) stated that the anaemia of infancy due to sepsis is aregenerative or hypoplastic in type. Infection may depress the bone marrow in such a way as to delay the full maturation of the erythrocytes or possibly prevent the bone marrow from delivering the red cells to the blood stream. In some of our cases the development of an infection, during the period of recovery, has clearly been responsible for preventing the expected haemoglobin rise or has obviously depressed the haemoglobin level during the active phase of the infection. At the same time, there may be a greater demand for folic acid or its full utilisation may be prevented.

Iron deficiency. — Smith (1948) described the anaemia of chronic infection as normocytic, normochromic and iron deficient. Iron deficiency as the cause of the anaemia of kwashiorkor has not been accepted by us in the past because of the high iron content of the maize diet of our patients and the presence of haemosiderin particles in the bone marrow. Since Trowell and Simpkiss (1957) reported good responses in infants after intramuscular iron we have reconsidered our views and are, at present, investigating more fully the haemosiderin content of the bone marrow 28 days after full treatment with and without extra iron. It is unlikely that acute infections to which our patients are particularly subject alone prevent full recovery and other abnormalities must also be considered, particularly protein synthesis enzyme formation and other deficiencies.

Protein deficiency anaemia. — The exact role of protein lack in the aetiology of anaemia in kwashiorkor is most difficult to assess. Some early workers (Altmann and Murray, 1948; Adams, 1954; Trowell *et al.*, 1954) regarded this as a possible primary cause, while Woodruff (1955) appeared to prove this in his series of patients who would not respond to vitamin B_{12}, iron or folic acid, but who did improve very gradually when the diet was supplemented with casein. Foy and Kondi (1957), on the other hand, state that the type of anaemia in kwashiorkor is not due to a specific deficiency and that dietary supplements have no effect upon it unless appropriate haematinics are also given. I find the haematological response is tardy despite high protein intake together with haematinics. This may perhaps be due to the large amount of protein used for growth and recovery leaving insufficient available for the construction and full maturation of the red cells.

Aschkenasy (1957), experimenting with rats, produced a protein deficiency anaemia which was hypochromic and microcytic in marked contrast to our patients whose anaemia is normocytic and normochromic. The bone marrows, however, in his rats were similar to those in our patients being hyperplastic and normoblastic. My difficulty is to explain the very slow rise in the haemoglobin level despite treatment with ascorbic acid, folic acid, antibiotics and high protein feeding and despite a rise of total serum protein within two weeks of initiating treatment. It is pertinent to recall that in 1951 Gillman and Gillman wrote: «If the diet is deficient in many essential nutriments, it is impossible to dogmatise as to the specific deficiency which is responsible for a particular anaemia.» If protein deficiency alone was the principal limiting factor, a parallel rise in haemoglobin might be expected, but haemoglobin synthesis lags far behind the excellent rise in serum proteins, which is achieved by the fourteenth day of treatment (Fig. 1).

Future therapy. — In Durban our patients are believed to be deficient in protein and folic acid but remain anaemic even after adequate therapy. Because kwashiorkor is a general metabolic disease, it may be impossible for the severely malnourished patient to synthesise sufficient haemoglobin during the first 28 days of treatment. In this series, however, a few of the milder cases have been able to do so. It may be asking too much of our severely malnourished infants to synthesise more haemoglobin than they do during the first four weeks of recovery; they may be able to restore their haemoglobin completely in time even without additional haematinics. However, it is also possible that iron, cobalt, copper or some other substances may become deficient as a result of new processes initiated during convalescence and thus limit full haematological recovery. Coles (1954) reported a good response to cobalt sulphate in 31 anaemic infants, while Tevetoglu (1956), more recently, recorded similar results using a cobalt-iron mixture. Further studies along these lines using multiple therapeutic agents appear to be indicated in the attempt to cure the anaemia of kwashiorkor more expeditiously.

SUMMARY

1. The two principal types of anaemia in kwashiorkor, as seen in Durban, are described.

2. Our routine treatment is recorded.

3. The results obtained after treating 23 cases of anaemia in kwashiorkor are summarised and briefly dicussed.

4. The pattern of the haemoglobin fall and rise over four weeks of treatment is shown graphically and attention is drawn to the marked delay in re-establishing normal values.

5. No single factor has been discovered as the cause of the anaemia.

6. Further studies using extra iron are now in progress and it is suggested that treatment with many haematinics may ultimately be the therapy of choice.

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HEPATIC VENO-OCCLUSIVE DISEASE: ITS WORLDWIDE DISTRIBUTION IN MAN AND ANIMALS

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Hepatic veno-occlusive disease is predominantly seen in children aged 2 to 5 years, although adults are also affected. Three clinical stages have been defined: an acute stage, characterized by ascites and hepatomegaly; the subacute stage; and the chronic stage, in which there is cirrhosis of the liver (1).

Histologically, the essential finding in the acute stage is a blockage of the medium-sized and smaller hepatic veins by a subendothelial swelling of the intimal tissues. This swelling is apparently due to oedema but later becomes collagenous. This intimal occlusion produces a block centrilobularly with distension of the tributary sinusoids so that macroscopically the appearance resembles that of a «nutmeg» liver cardiac congestion (1).

The subacute stage shows a persistence of centrilobular fibrosis (nonportal) and the chronic stage is that of a fully developed nonportal cirrhosis (1).

This disease has been reported in humans in the West Indies (1), South Africa (2, 3), and India (4), and is thought to be due to the ingestion of «bush teas» or food containing the pyrrolizidine group of alkaloids found in *Compositae* (genus *Senecio*) and the *Leguminosae* (genus *Crotalaria*) (5). There have also been reports from Germany (6), and suspected cases from the Middle East — Egypt and Israel (7). The disease has been produced experimentally in rats by the injection of monocrotaline, one of the pyrrolizidine group of alkaloids isolated from several species of *Crotalaria (Leguminosae)* (8). It has also been produced by experimental feeding of horses with *Senecio riddelli* (9), and in a cow with *Crotalaria fulva* (10).

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The disease has also been reported in field animals in the United Kingdom — cattle and horses after ingestion of ragwort (1, 11); in the United States — horses and cattle after ingestion of species of *Senecio (Compositae)* (9) and *Crotalaria (Leguminosae)* (13) respectively; from descriptions and photographs in the literature it would appear that veno-occlusive disease is found in horses and cattle in South Africa and in horses in Australia (13).

Considerable investigation has been carried out on the various diseases in animals caused by the ingestion of the above plants; such diseases are the walking disease of horses and cattle in Northwestern Nebraska, U.S.A. (14), dunsiekte or enzootic liver disease of equines in South Africa (15, 16, 17), walkabout disease in Northwestern Australia (18), Winton disease of New Zealand (18), and ragwort poisoning of horses and cattle in the United Kingdom (19). In these diseases various lesions of the liver, other than veno-occlusive disease, have persistently been described; they are centrilobular congestion with «lagoons of blood», haemorrhagic necrosis, cirrhosis, megalocytosis (20), endothelial hyperplasia, and neoplasia (21).

It would thus appear that hepatic veno-occlusive disease is only one aspect of the hepatotoxic properties of the pyrrolizidine group of alkaloids, which are causing disease all over the world.

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TROPICAL NUTRITION AND SOME DISORDERS OF THE EYE

H. A. P. C. OOMEN [1]

The eye is a useful indicator of nutritional disease. Many vulnerable tissues and structures are present in and around the eyeball. Some are accessible to the naked view, but if special methods are required, the scarce and busy practitioner of the tropics will not have the same observation facilities as his counterpart in prosperous regions. Therefore, less striking, but important signs may easily be overlooked. But even striking manifestations still may arise from unexpected corners, as proved, for instance, by the many cases of diabetic cataract, in whom Zuidema discovered a pancreatic necrosis.

I am not going to review the minor eye signs, which often are present in sufferers from malnutrition. I fervently hope that you do not expect from me to define what is a nutritional disease of the eye and what not. I want to concentrate on two eye affections, which nowadays seem to be specially prevalent in the tropics. Of both I would prefer to emphasize the general significance.

Of the first, *retrobulbar neuropathy*, I believe I still have to explain why I consider it as a nutritional disease. Of the second, xerophthalmia, this is superfluous. Both show close relations to deficiency signs in organs or tissues, elsewhere in the body. But in both the eye lesion dominates the clinical picture. Retrobulbar neuropathy is roughly an affection of adults, while xerophthalmia has a definite predilection for the small child. The former invalidates the victim to a serious degree, but seldom causes complete blindness. The latter causes blindness and even death in many cases, and may be the more frequent. The neural, perhaps better neurovascular, affection of the

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eye is apparently not so easy to cure and probably not simply to prevent. The epithelial lesion responds very readily to the missing nutrient, while ways of prevention at least seem obvious. Both urgently need a clearer definition of their clinical, pathological and epidemiological aspects. From the biochemical and experimental viewpoint a huge literature exists regarding vitamin A, but practically none as to the optic nerve affection.

Is retrobulbar neuropathy actually a nutritional disease? This recently has been denied by Degazon (1956), who proposes the term *tropical amblyopia*. He reviewed about 300 patients from Jamaica, the same locality from where early descriptions of the condition stem (Strachan, 1897; Scott, 1918). Part of this patients enjoyed a reasonable diet and there was no clear improvement on dietary therapy. They were out-patients from a free community. Now retrobulbar neuropathy has often been reported from closed communities, eating from a central kitchen. I don't think that the many doctors who experienced the disease in the widely dispersed Japanese internment camps during the last war have any doubt as to the nutritional nature. Retrobulbar neuropathy then behaved as one of the waves of nutritional disorders which successively struck the internees. Burning feet and camp eyes belonged to the first, puzzling manifestations; pellagra and hunger oedema, in some surroundings also beriberi, came later on worse diets, after more prolonged exposure. It is significant that the limited documentation on the subject mostly considers the early periods of internment, when diets were not yet too bad notably as to calories and protein. Few new cases developed in the huge civilian camp at Tjimahi (Java) and some even improved during the last period (1 ½ year), when diet was worst. We doctors were very scared at the first appearance of the eye lesion, but in the long run pellagra and hunger oedema proved to be more dangerous foes. The diet on which both «camp eyes» and burning feet developed (Spillane & Scott, 1945; Hobbs and Forbes, 1946; de Wijn, 1947; Smitskamp, 1947) was about the same and sometimes more copious than the average intake of the majority of the Javanese population. At the time of the peak incidence of new cases, emaciation was not yet a serious problem. Burning feet and camp eyes behaved as separate clinical entities. While roughly in 1/3 of the cases both conditions overlapped each other, the majority of the stricken had either burning feet or camp eyes. The same has been reported for camp eyes and the syndrome of nerve deafness and ataxia (Spillane & Scott, 1945; de Raadt, 1947; Cruickshank, 1956). The eye affection was dissociated as well as to onset as to the patient, from either pellagra, beriberi or hunger oedema.

Camp conditions in S.E. Asia resembled a huge nutritional experiment of 3 ½ years duration, which produced only a limited amount of documentation. I believe that they permit a few though important conclusions:

1. Retrobulbar neuropathy is not necessarily associated with either pellagra, beriberi, hunger oedema or burning feet. It is a separate clinical entity.

2. The affection is closely dependent on dietary conditions, but it is not a specifically tropical condition.

3. The neuropathy is however not a clinical attribute of extreme dietary limitations; the latter possibly even prevent the development of the syndrome.

4. No single vitamin has proved to be able to reverse the condition. But additional animal protein (especially eggs) gave probably therapeutic results in the active stage.

Bietti (1950), reviewing the relations between nutrition and the condition of the eye, stated that optic nerve lesions were perhaps the most intricate and obscure he had encountered. The explanation could be that it is a conditioned deficiency, which apparently becomes manifest at a definite dietary level. Cruickshank (1956), who considered retrobulbar neuropathy, ataxia and nerve deafness as allied symptoms, suggested two possibilities: 1) deficiency of substances essential for nerve metabolism; 2) dietary factors of a toxic nature, either extrinsic or intrinsic.

The nutritional aetiology of *xerophthalmia* is more distinct. Still obscure points remain and prevention of this dreadful condition has not yet made much headway in several tropical regions. It was a current subject of clinical attention in the European ophthalmologic journals in the last half of the preceding century. About 1900 it was still prevalent in Japan (Mori, 1904). War limitations produced a few hundreds of cases in Denmark during and after the first world war (Blegvad, 1924). Since then it seems to have vanished as a public health problem from temperate countries. Such is not the case in some S.E. Asian countries. I quote an example of over 10,000 cases observed in a single eye hospital in central Java during a period of 20 years (Oomen, 1957).

Though the affected are from every age, even from birth on, there is a definite predilection for the toddler period, with a peak incidence in the third and fourth year. With increase in age, the symptoms often become less severe. In the schoolchild, for instance, Bitot spots are the more common sign. In breastfed babies, the aetiology must be different, because in communities which predominantly use vegetarian foods, the suckling is the only one, who finds the preformed vitamin in this diet. The young, weaned child must derive his vitamin A from the carotene in his food. If this is lacking, the door is open to epithelial damage. But sometimes carotene is consumed in apparently sufficient quantities. Then a disturbance of conversion or resorption must play a role.

Not only vitamin A or its precursor are often lacking in the diet of the affected. Common is also a deficiency in protein intake (Oomen, 1957) and in fat (Roels, 1958). At least in Indonesia there is a close relation between kwashiorkor and xerophthalmia (Oomen, 1954). Perhaps this is the reason why xerophthalmia there is so frequent and often so severe. There is also a close link between infectious disease in childhood, either acute or chronic, in so far that xerophthalmia often develops in its wake. This may be considered as further proof of the vulnerability of the situation. But as similar conditions threaten the small child also in other tropical regions, it is remarkable that xerophthalmia is seldom considered as an important disease or admitted to the sanctuary of tropical textbooks. A complicating factor is that the terms *xerophthalmia, xerosis* and *keratomalacia* may not indicate the same condition in different regions and may lead to confusion.

The cure of xerophthalmia with vitamin A is easy, provided that the condition is still reversible. But the emergency is not always recognized, and then irreparable harm may result. The prevention is more difficult as the affection is particularly prevalent in countries without a cow's milk tradition. Other animal foods then have to provide the vitamin. If even not this is possible, not only carotene sources should be present in the diet, but also enough protein and fat.

SUMMARY

The two most important nutritional disorders of the eye in the tropics probably are xerophthalmia and retrobulbar neuropathy. As a public health problem under normal conditions, they nowadays have vanished from temperate countries. Their nature and significance are discussed. A plea is made for a more accurate and efficient approach.

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GENERAL DISCUSSION

Dr. B. S. Platt (U.K.) — I want to draw attention to some recent work (Lutz & Platt, 1958) on the composition of human milk in mothers who are malnourished and normally nourished. The total protein of human milk falls only slightly in protein malnutrition as compared with the normal, but striking differences are found in the ratio of the curd to whey proteins (C:W ratio). In English mothers the value for this ratio is about 1.0 and the scatter is small. In malnourished mothers, e.g. a group of African women in the Belgian Congo and some Indiam mothers in Calcutta, a C:W ratio of about 1.3 with values as high as 2.4 is found. These differences in the C:W ratio for milk correlate well with the values for urea nitrogen:total nitrogen in the urine and with the plasma albumin levels — with high C:W ratios, low values are found for urinary urea nitrogen and serum albumin (see Platt, 1958, Fig. 5).

It would seem as if the tendency to maintain a nearly constant concentration of protein in human milk is achieved by increased synthesis of casein, most if not all of which occurs in the mammary gland itself. The output in the milk of whey proteins is often absolutely and relatively low and it is suspected that it reflects the state of nutrition of the extra mammary development of the mother where some at least of the whey proteins may be synthesised. It may be noted that this work, directed towards the study of the differences between the curd and whey fractions of milk, was prompted by observations on the infant rat stomach, from which it had appeared that the curd and whey fractions had a different fate and it was suspected, as the above evidence tends to show, that the proteins of these two main fractions may also have a different origin in the mother. On electrophoresis of whey, proteins can be separated into three main fractions and it is interesting that the proportions of two of these alter when there is an alteration, as in the Indian mothers, of the ratio of albumin to globulin in the blood plasma.

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DR. MCDONALD (Africa) — In connection with the effect of nutrition of the eye, I should like to report the findings of Dr. Blumenthal in South Africa and Dr. McLaren in East Africa on the condition known as spontaneous prolapse of the iris.

Blumenthal first described in 1950 a malnutritional keratitis in which one feature of the condition was a spontaneous prolapse of the iris seen mainly in the post-weaning stage with a slight increase in incidence at puberty.

The mechanism suggested by Blumenthal as underlying the condition was an alteration in the stroma of the cornea followed by a localized bulging of Descemets membrane. This membrane may rupture and thus enable the iris to enter the substantia propria of the cornea and cause a localized swelling of the epithelium on the anterior surface of the eye.

Blumenthal's assumption that this is the mechanism rests on the following evidence:

1. The epithelium of the cornea is frequently seen intact in this condition.

2. In several cases the corneal epithelium was ruptured and histologically it could be seen that intact Descemets membrane covered the knuckle of prolapsed iris.

The aetiology of the condition is, according to Blumenthal, entirely nutritional and infection is only secondary. He has not seen a case of prolapsed iris in an emaciated child, but only in children that are fat and podgy, and he believes that a predominantly carbo hydrate diet without vitamins is dangerous from the ophthalmological point of view.

Further and more detailed evidence that the prolapsed iris is nutritional in origin lies in the fact that the addition of vitamin B complex to the child's original diet, and with no local treatment, results in the deeper corneal component being arrested. The inflamed surface of the eye is cleared by the addition of vitamin A to the diet.

McLaren, in his preliminary communication, has noted this eye condition in East Africa and, though agreeing with Blumenthal on the clinical condition, differs from him in respect to mechanism and causation.

McLaren, in a preliminary communication has noted an absence of relationship between the prolapsed iris and what he calls protein malnutrition and furthermore has seen cases in marasmic infants as well as in apparently well nourished children and otherwise healthy adults.

Injury plays no part and McLaren confirms Blumenthal's findings that the corneal epithelium is unaffected. McLaren, however, believes that the primary lesion lies in the endothelium and *not* in the stroma of the cornea. The endothelial change he describes as consisting of bubble patches of degeneration and later the overlying stroma becomes infiltrated with cells and blood vessels.

McLaren cannot accept that avitaminosis B plays any part in the causation of the condition and suggests the following hypothesis: The prolapsed iris results from a purely endothelial change in the cornea and this endothelial change is the result of a deficiency of fatty acids. This hypothesis, he believes, is suggested by experimental work in rats in which essential fatty acid deficiency leads to changes in the connective tissue supporting the endothelium of capillaries.

This hypothesis is compatible with the excellent general nutrition of his cases and the low fat and fatty acid intake of very young African children.

Deficiency of pyridoxine, however, may lead to prolapsed iris due to the role of this vitamin in the conversion of linoleic to arachidonic acid, even in the presence of an adequate intake of essential fatty acid as is found in diets of most African adults.

Dr. Woodruff (U.K.) — The emphasis which Dr. Walt has laid on the occurrence of anaemia in patients with kwashiorkor who have no coincident helminthic or malarial infection is welcome, for some workers have recently tended to regard anaemia in these cases as being caused by such coincident infection. The observation that megaloblastosis may be corrected in these cases without improvement of the anaemia is also of interest, for Bell, working in the Gambia, has reported the same thing. Lack of protein may not be the only factor in the causation of these anaemias, but the evidence is that it is the most important one. There is, for example, ample experimental evidence for protein deficiency as a cause of anaemia. Whipple and Madden (1949) developed the concept of «a large protein pool including plasma proteins [...] The contributions to this pool derive largely from the liver [...] From this protein pool may be derived haemoglobin, plasma protein or cell protein».

A further link in the chain whereby protein malnutrition may lead to anaemia had been provided by Nizet and Robschiet-Robbins (1950), who, in experimental work with dogs, have shown the rate at which reticulocytes ripen to depend on the constituents of the plasma. They have concluded that the red blood cell at the reticulocyte stage is able to incorporate plasma protein into itself or to synthesise plasma proteins from amino acids. Thus, if the plasma proteins are abnormal or deficient, some disturbance may take place in the ripening of reticulocytes and therefore in the formation of the mature red blood cells. Woodruff (1955) showed that in children suffering from kwashiorkor the ripening of their reticulocytes was delayed.

Further experimental evidence of the parts that protein deficiency and liver damage may play in the production of anaemia is obtained from the work demonstrated by Prof. B. S. Platt at this Congress.

Whipple and Madden (1949) have stated that contributions to the plasma protein derive largely from the liver. In view of this, and of the part that these plasma proteins may play in haemoglobin synthesis, the widespread high incidence of liver damage among tropical peoples almost certainly plays some part in the causation of the common anaemias there. It has to be borne in mind that the serum albumin values of patients suffering from protein malnutrition may be as low as 0.2 g percent.

It is difficult to recognise anaemia caused by protein malnutrition because it seldom presents in pure form in man, but in experimental animals it is of simple normochromic, normocytic type. In man concomitant iron deficiency often imposes its own characteristics upon it and leads to the anaemia present being considered to be the result of iron deficiency only. If, however, liver damage is associated with the anaemia of protein malnutrition, then target cells may be found in the peripheral blood and the red cells generally will be broader and thinner than normal. Cells of this type were described by Larsen (1948) and physicians and pathologists who have dealt with patients with cirrhosis of the liver are familar with them. Himsworth (1950) has reported similar broad and thin cells in rats suffering from methionine deficiency and liver damage and Woodruff (1955) has found them to occur in children suffering from anaemia and kwashiorkor, in pregnant women suffering from anaemia and protein malnutrition and in men and non-pregnant women similarly affected. These cells may give the blood the appearance of macrocytosis when a blood film only is examined and they have certainly been the cause of confusion between anaemia associated with liver damage and that due to deficiency of specific haemopoietic factors.

Doubts on the part played by protein malnutrition in the production of anaemia may be based on the poor therapeutic response obtained in these anaemias following administration of protein. It must, however, be remembered that the functional capacity of the liver to convert dietary protein into plasma protein suitable for haemoglobin synthesis may be impaired and such functional impairment of the liver may be irreversible or may be reversed only slowly and by prolonged treatment over two or three or even more months.

In view of these considerations, it appears to be important to provide liberal amounts of protein over a prolonged period to patients suffering from anaemia with kwashiorkor or protein malnutrition.

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Prof. J. Sénécal (Dakar — A.O.F.) — Pour mesurer la perte des réserves protéiques nous voudrions ajouter aux procédés employés par John Waterlow deux techniques qui nous paraissent intéressantes. La première complète en quelque sorte la technique d'analyse des tissus. C'est l'examen au microscope électronique d'un fragment de foie prélevé par biopsie.

Les projections illustrent ce qu'on peut en atteindre.

Les 3 premiers vues ont été prises à des grossissements différents chez un enfant atteint de kwashiorkor à son arrivée. Elles montrent essentiellement la graisse qui infiltre tout le parenchyme et surtout l'atteinte et même la destruction de mitochondries. Celles-ci deviennent claires, floues, perdent leur striation et même se lysent. Les trois vues suivantes montrent l'aspect du foie chez le même enfant après 11 jours de traitement. On note la disparition de la graisse, l'apparition de grains fins d'acide desoxyribonucléique, les mitochondries deviennent denses, les crêtes sont bien visibles et des complexes mitochondries-ergastoplasme se forment.

Au faible grossissement on voit que cet ergastoplasme se developpe volontiers au contact du noyau et de la membrane.

Une deuxième manière d'aprécier la perte ou la restauration des protéines est tirée des enseignements du traitement du kwashiorkor. L'expérience nous a montré que pour éviter les rechutes il fallait poursuivre pendant plusieurs semaines le régime hyperprotidique, qui pour nous comporte des doses très élevées de protéines animales (8 g à 10 g par kilogramme de poids et par jour). En 8 jours le taux de protéines remonte et même dépasse le chiffre de 7 g pour-cent. Mais ces protéines circulantes ne représentent qu'une faible partie de protéines corporelles (20 %) et il est nécessaire de poursuivre le régime pendant plusieurs semaines pour guérir completement l'enfant.

Ceci a été démontré par Dr. Mayer du Congo Belge, qui, mettant des enfants à un régime completement privé de protéines, a obtenu des résultats différents chez l'enfant normal et chez l'enfant atteint de kwashiorkor. Chez le premier le taux de protéines sanguines demeure stable pendant 15 jours; chez l'enfant convalescent, dès le deuxième jour, on assiste à une chute de la protéinemie et particulièrement de l'albuminemie.

Ces deux méthodes ne sont evidemment pas de pratique courante et il reste encore à découvrir le test qui permettrait de faire le diagnostic des formes frustes.

À ce sujet nous rappelons qu'à la conférence sur la nutrition de Luanda (Octobre 1956) nous avons, avec M^{me} Aubry, présenté un travail signalant la fréquence des formes frustes et insistant sur la valeur des variations des globulines α et β.

Dr. J. H. Walters (U.K.) — In 1945 Drs. Rossiter, Lehmann and myself studied the regeneration of the blood on refeeding Indian soldiers who had suffered prolonged and severe protein malnutrition during 3 ½ years captivity in Japanese prison camps. We carried out serial estimations of the concentration of red cells, haemoglobin and plasma protein together with the blood volume by the Evans Blue technique, and thus calculated total figures for these three elements. The plasma protein regenerated much more rapidly than did the red cells and haemoglobin, and the plasma volume rose rapidly to an abnormally high level, thereafter subsiding slowly to normal as the total red cell volume rose. In view of this dilution effect, it may be misleading to draw conclusions from figures representing concentrations of these three elements during recovery from protein malnutrition: 28 days may be too short a period for the full regeneration of the blood to be completed.

Prof. K. Hill (U.K.) — We believe that V.O.D. is caused by a poison — one of the alkaloids of the pyrrolizidine group. Other factors may play a part. Most humans affected have a low dietary protein and low serum albumin; also animals affected eat *Senecio* and *Crotalaria* plants in times of poor grass production.

Further we have found that V.O.D. appears to be much more evident when the experimental animal is infected. This in line with the observations of Rhodes, who found that at least 50 % of cases of V.O.D. were preceded by an acute infection, either measles or whooping cough, or respiratory tract infection. «Bush teas», of course, are often given as medicine to counteract infection!

2ND SESSION: **Metabolism in tropical malnutrition**

Wednesday, September 10th — 11:00 to 12:30 a.m. Room B

The meeting opened under the chairmanship of Dr. H. A. P. Oomen.

The Rapporteur was Dr. L. MacDonald and the Secretary was Dr. Carlos dos Santos Reis.

The following papers were presented and discussed.

DETERMINATION DU BESOIN MINIMUM EN Ca CHEZ LE BANTOU À L'AIDE DU Ca 45

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et
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Dans une série de travaux antérieurs nous avons établi, par la méthode classique des bilans, le besoin minimum en Ca chez un grand nombre d'indigènes de Kwango (Congo Belge), dans diverses conditions. Chez l'enfant Africain «normal» de cette région nous avons trouvé une valeur moyenne de 12 mg/kg/24 h. — Holemans et Lambrechts (4). Pour des jeunes femmes bien nourries un besoin minimum moyen de 4 mg/kg/24 h. a été constaté et pour des hommes «normaux» du milieu coutumier environ 10 mg/kg/24 h. — Lambrechts et Holemans (7). Dans le kwashiorkor, le besoin minimum est un peu plus élevé que chez l'individu normal — Holemans et Lambrechts (5) — aux environs de 20 mg/kg/24 h. Enfin pour la femme allaitante, nous avons signalé — Holemans, Lambrechts, Hubin et Martin (6) — un besoin de 12 mg/kg/24 h. environ de Ca.

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 194-200, 1959

Comme très peu de travaux ont été exécutés dans les zones tropicales et subtropicales à faible consommation de Ca sur le point précis du besoin minimum d'une part et que Hegsted (2, 3) a critiqué récemment la technique des bilans pour l'établissement de cette donnée d'autre part, nous avons pensé qu'il était utile de confirmer nos résultats par l'utilisation de Ca 45. Jusqu'à présent, nous n'avons pu appliquer cette méthode au Kwango (Congo Belge) qu'aux cas de jeunes garçons et de femmes allaitantes.

TECHNIQUES

Une faible dose de Ca 45, de l'ordre de $^1/_{25}$ à $^1/_{50}$ de la dose de tolérance indiquée par Martinelli (9), a été administrée per os à 8 jeunes garçons et 5 femmes allaitantes suivant deux modalités.

Groupe I. — 4 garçons âgés de 13 ans environ et d'un poids moyen de 25 kg ont reçu chacun, pendant 3 jours, une dose totale de 0,03 microcurie Ca 45 par kilogramme intimement mélangé au régime coutumier constitué par de la pâte de manioc à volonté (500 g à 1.000 g) enrichi de poisson séché (150 g environ). Pendant cette période et les 7 jours suivants le régime, les selles et les urines sont récoltés quantitativement et analysés pour Ca stable, Ca 45 et P.

Groupe II. — 4 autres garçons approximativement du même âge et du même poids ont reçu au début d'une période de bilan de 10 jours une dose totale unique de Ca 45 du même ordre que les précédentes (0,02 à 0,04 microcurie par kilogramme). Le radiocalcium a été ajouté au premier repas de la période de bilan. Régime, selles et urines sont analysés comme pour le groupe I.

Groupe III. — 5 femmes en lactation depuis 8 à 24 mois, et pesant en moyenne 40,3 kg, ont reçu, comme les garçons du groupe I, une dose totale de 0,03 microcurie par kilogramme de Ca 45 répartie sur 3 jours et mélangée au régime; Ca stable et radioactif, P. et N. sont dosés pendant 10 jours dans les ingesta et excréta. Les résultats pour Ca stable, P. et N. ont été publiés — Holemans, Lambrechts, Hubin, Martin (6).

Le groupe II reçoit le même régime de base de pâte de manioc et de poisson séché que le groupe I avec un supplément d'arachides grillées et de légumes verts indigènes. Le groupe III a le même régime *ad libitum* que le groupe I. L'alimentation de ces 3 séries est qualitativement très semblable à celle du milieu coutumier: elle contient donc peu de Ca (± 15 mg/kg/24 h.).

Le besoin minimum en Ca a été déterminé par le Ca 45 à l'aide de deux procédés différents: *a*) pour les groupes I et III, le Ca fécal endogène est calculé par une méthode originale — Lambrechts, Reginster, Hubin (8) — consistant essentiellement en la comparaison des activités spécifiques du Ca alimentaire et du Ca fécal; *b*) pour le groupe II, la technique de Monte Blau et coll. est utilisée (1).

RÉSULTATS

Il serait trop long d'envisager chaque sujet en particulier; il est plus commode d'établir une moyenne de résultats par groupe considéré.

Groupe I. — Poids moyen des sujets: 25,5 kg.

Calcium ingéré: 205 mg/jour = 8 mg/kg/24 h.

Activité spécifique moyenne du Ca alimentaire: 16,2.

Activité spécifique du Ca fécal des 3 premiers jours: 3,2.

Ca fécal total en moyenne: 253 mg/24 h. ou 10 mg/kg/24 h.

$$\text{Ca fécal endogène/24 h.} = \frac{\text{act. spéc. Ca selles}}{\text{act. spéc. Ca aliment.}} \times \text{Ca fécal total} = \frac{3{,}2}{16{,}2} \times 253 \text{ mg} = 51 \text{ mg.}$$

Ca fécal endogène par 24 h. = 253 mg — 51 mg = 202 mg ou 8 mg/kg/24 h.

L'absorption intestinale est de 205 mg — 51 mg = 154 mg/24 h. ou 75 %.

Ca urinaire mg/kg/24 h. = 102,5 : 25,5 = 4 mg.

Le besoin minimum théorique correspondant à la somme des pertes fécales et urinaires est donc de 8 mg + 4 mg = 12 mg. La couverture de ce besoin par l'alimentation doit tenir compte de l'absorption intestinale. Dans le cas présent cette valeur étant de 75 %, le besoin alimentaire réel sera de $8 \text{ mg} + 4 \text{ mg} \times \frac{100}{75} = 16 \text{ mg/kg/24 h.}$

Pendant cette période qui a servi à la détermination du Ca fécal endogène, le bilan de Ca stable a été nettement négatif; en d'autres termes, l'ingestion de 8 mg/kg/24 h. n'a pas été suffisante pour couvrir le besoin minimum alimentaire réel que le calcul a établi à 16 mg/kg/24 h.

Groupe II. — Quand une dose unique de 0,03 microcurie de Ca 45 est administrée *per os* et que le Ca 45 et le Ca stable sont dosés dans les selles pendant les jours suivants, l'activité spécifique fécale décroît rapidement pendant 3 jours, correspondant à l'élimination du Ca 45 non absorbé, puis beaucoup plus lentement à partir du 4^{me} jour. À ce moment, le Ca 45 des selles provient uniquement des sucs digestifs et marque donc le Ca endogène. L'activité spécifique du Ca des sécrétions digestives est la même que celle du sang et de l'urine.

Chez les sujets en expérience, l'activité spécifique du Ca fécal du 6^{me} et du 7^{me} jour est la même que l'activité spécifique du Ca urinaire; en d'autres termes, le Ca fécal est constitué uniquement par du Ca endogène.

Poids moyen des sujets: 25,7 kg.

Ca urinaire moyenne/jour = 51 mg ou 2 mg/kg/24 h.

Activité spécifique = 0,16.

Ca fécal endogène/jour = 194 mg ou 7,7 mg/kg/24 h.

Activité spécifique = 0,17.

Besoin minimum théorique = 7,7 mg + 2 mg = 9,7 mg/kg/24 h.

Puisque l'absorption du Ca alimentaire est totale chez ces sujets, le besoin alimentaire réel est identique au besoin minimum théorique.

Pendant cette période, l'ingestion moyenne de Ca stable est de 24 mg/kg/24 h. et le bilan est positif malgré la présence de P. phytique dans le régime.

Groupe III. — Poids moyen des sujets: 40,3 kg.

Ca ingéré: 464 mg/jour ou 10,1 mg/kg/24 h. en moyenne.

Activité spécifique moyenne du Ca alimentaire: 7,2.

Activité spécifique moyenne du Ca fécal des 3 premiers jours: 1,6.

Ca fécal total en moyenne par jour = 300 mg.

$$\text{Ca fécal exogène/24 h.} = \frac{\text{act. spéc. Ca selles.}}{\text{act. spéc. Ca aliment.}} \times \text{Ca fécal total} = \frac{1{,}6}{7{,}2} \times 300 = 66 \text{ mg.}$$

Ca fécal endogène/24 h. = 300 mg — 66 mg = 234 mg ou 5,85 mg/kg/24 h.

L'absorption intestinale est de 464 mg — 66 mg = 398 mg ou 85,7 %.

Ca urinaire moyen = 80,5:40,3 = 2 mg/kg/24 h.

Le besoin minimum théorique = 5,8 mg + 2 mg = 7,8 mg/24 h.

Comme l'absorption intestinale est de 85 %, le besoin minimum alimentaire réel est de 7,8 mg $\times \frac{100}{85} =$ 9,1 mg/kg/24 h.

Pendant cette période le bilan de Ca stable est positif.

En analysant les selles et les urines pendant les jours suivants l'administration du Ca 45, il a été possible de calculer le Ca fécal endogène comme pour le groupe II: le jour où l'activité spécifique des selles est égale à l'activité spécifique des urines, le Ca fécal total est de 227 mg en moyenne. A ce moment il s'agit uniquement de Ca fécal endogène correspondant donc à 5,6 mg/kg/24 h. On retrouve donc de cette façon la même valeur pour le besoin minimum que celle trouvée par l'autre méthode, puisque 5,6 mg + 2,3 mg (urine) = = 7,9 mg/kg/24 h. Pendant ce temps le bilan de Ca stable est négatif.

DISCUSSION ET CONCLUSIONS

On pourrait objecter que l'addition de Ca 45 à l'alimentation ne constitue pas véritablement un étiquetage du Ca du régime. La remarque est judicieuse du point de vue théorique, mais l'aspect technique et les résultats font admettre qu'en pratique l'échange des atomes de Ca 45 et de Ca stable s'est fait d'une manière adéquate et qu'une activité spécifique du Ca exogène peut être considérée comme valable: d'une part le Ca 45 est mélangé intimement à la grande masse de la pâte de manioc contenant la presque totalité du Ca alimentaire et, d'autre part, les résultats par notre méthode de calcul du Ca endogène et par celle de Monte Blau sont pratiquement identiques.

Les résultats mentionnés ci-dessus pour le besoin minimum, et obtenus par l'étude de 130 jours de bilan de Ca chez 13 sujets à l'aide du Ca 45, concordent suffisamment avec les valeurs obtenues pour les deux catégories d'individus (garçons et femmes) à l'aide du Ca stable. Le bilan classique exprimant la relation entre ingestion et rétention renseigne sur la valeur globale du besoin minimum, c'est-à-dire le besoin minimum alimentaire; l'établissement du Ca fécal endogène n'en constitue qu'un élément, auquel il faut ajouter le Ca urinaire, et leur somme doit être affectée d'un coéficient correspondant à la non-utilisation digestive. Il faut en outre remarquer qu'on obtient des valeurs identiques pour le Ca fécal endogène, qu'on le calcule soit par la méthode de Lambrechts et coll. soit par celle de Monte Blau.

Pour le groupe des jeunes garçons, les valeurs suivantes ont été trouvées ci-dessus pour le besoin minimum global: 16 mg, 13 mg, 9,7 mg, ce qui représente une valeur de 12,9 en moyenne contre 12 mg/kg/24 h. par la méthode classique des bilans de Ca stable.

Pour le groupe des femmes allaitantes, les valeurs sont de 9,1 mg et 7,9 mg, alors que le besoin minimum établi par les bilans de Ca stable est de 10 mg/kg/24 h. environ, la perte par le lait non comptée.

Il est intéressant de noter que le besoin minimum de Ca peut être déterminé à l'aide de Ca 45 que le bilan de Ca stable soit positif ou négatif.

Une autre conclusion importante peut être tirée de l'identité des résultats obtenus par la méthode des bilans et la technique au Ca 45: contrairement aux critiques de Hegsted (2, 3), la méthode des bilans conserve toute sa valeur pour l'établissement du besoin minimum en Ca, tout au moins dans les conditions expérimentales où nous l'avons recherché, c'est-à-dire aux ingestions basses de Ca.

RÉSUMÉ

Le besoin minimum en Ca a été déterminé chez 8 grands garçons et 5 femmes allaitantes du Kwango (Congo Belge) en ajoutant une faible dose de Ca 45 au régime coutumier pendant un bilan de Ca de 10 jours. Le Ca fécal endogène a été déterminé par 2 techniques différentes; en ajoutant à cette valeur la perte urinaire de Ca et en tenant compte de la grandeur de l'absorption digestive de Ca, on trouve le besoin minimum réel, qui est très voisin de la valeur obtenue à partir de la méthode classique des bilans de Ca stable, valeurs que nous avons signalées dans des travaux antérieurs.

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DETECTION OF MALNUTRITION BY A MODIFIED A.C.H. INDEX[1]

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Many somatometric methods and indices for judging the state of nutrition of children and adult persons have been devised in earlier years. The purpose of these methods is to indicate the influence of the diet on the rate of body growth and on the general state of the human body. Above all the child's development and state of nutrition was of special interest in this field. But in practice nearly all these methods have proved unsatisfactory, with the eventual exception of only one — the A.C.H. index by Franzen and Palmer in 1934. The results obtained by this test from children in tropical regions (India, South and East Africa), however, were rather contradictory. As in the years after the second world war there had been no further work relating to this index, the ratio between detection of malnutrition by the A.C.H. index and by clinical examination was studied by us once more.

In cooperation of the Tropeninstitut of Hamburg and the Instituto de Nutrición de la Universidad del Atlántico in Barranquilla, children 6 to 14 years of age were examined in the coastal regions of Colombia, where numerous races and miscegenations of all colours are to be found.

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[2] Held by G. Fuhrmann.

The A.C.H. index (arm-chest-*h*ip index) is based on the measuring of the circumference of the right arm at the highest point of the biceps with the forearm flexed and relaxed, the anterior-posterior diameter of the chest during inspiration and expiration, and the width of the hip from trochanter to trochanter. The A.C.H. chart of Franzen indicates the minimum difference between the sums of the arm girth and chest girth for the known hip width of the child. If this difference is below the figure on the chart, the child is selected as

TABLE 1 — *The A.C.H. chart of Franzen and Palmer*

Boys		Girls
Minimum difference between arm girth and chest depth (centimeters)	Width of hips (centimeters)	Minimum difference between arm girth and chest depth (centimeters)
0.0	< 20.0	0.5
0.0	20.0–20.4	1.1
0.4	20.5–20.9	1.6
1.0	21.0–21.4	2.1
1.6	21.5–21.9	2.6
2.2	22.0–22.4	3.0
2.7	22.5–22.9	3.4
3.2	23.0–23.4	3.8
3.6	23.5–23.9	4.2
4.2	24.0–24.4	4.5
4.7	24.5–24.9	4.8
5.1	25.0–25.4	5.1
5.6	25.5–25.9	5.4
6.0	26.0–26.4	5.6
6.3	26.5–26.9	5.8
6.7	27.0–27.4	6.0
7.0	27.5–27.9	6.1
7.3	28.0–28.4	6.2
7.6	28.5–28.9	6.3
7.9	> 29.0	6.4

suffering from malnutrition. According to the authors, the index shall identify children «with small amounts of musculature and fatty tissue relative to body build».

In our teamwork we carried out this set of measurements including height and weight, and at the same time the state of nutrition of the child was assessed clinically by an experienced paediatrician.

The results obtained were as follows: Totally examined by clinical inspection were 1,147 boys and girls — white and coloured — from 5 to 15 years, and by somatometric measures 865 children from 6 to 14 years of age. The clinical examination was carried out by means of a pattern containing all the data necessary for this purpose. The state of nutrition was given as «good», «fair», and «bad».

It could be stated that the arm girth as well as the body weight were considerably greater in the group of well-nourished children than among those showing a normal state of nutrition, while the other data — chest girth, hip width, body height — differed in these groups only slightly. The same occurs when comparing the groups «not selected» (containing the children classified as «good» and «fair») with the children of the group classified as malnourished. Selected as suffering from malnutrition by clinical inspection were 11.3 % of all children. From this number 42 % were also selected by the A.C.H. index, whereas from the children not selected by clinical examination another 10 % were selected by the A.C.H. index. Obviously these results were rather unsatisfactory; therefore we attempted to convert the A.C.H. index a more suitable form.

The A.C.H. index of Franzen and Palmer is nearly 30 years old, and we may assume that the processes of acceleration in body growth and development we can observe everywhere on the globe might be responsible for the unsatisfactory validity of the original A.C.H. chart. As these acceleration processes mainly occur in the later years of body growth, we subdivided the A.C.H. pattern into 3 groups of age, namely 6 to 8, 9 to 11, and 12 to 14 years, and, by statistical treatment, we could work out a new optimal correlation between clinical malnutrition and the various somatometric measurements. A further subdivision into yearly age groups did not improve this classification, nor did so a subdivision into only two age groups. Thus, the application of this modified A.C.H. chart yielded a great improvement in the results now obtained.

Table 2 — *Modified A.C.H. chart*

Boys			Width of hips (centimeters)	Girls		
Minimum difference between arm girth and chest depth (centimeters)				Minimum difference between arm girth and chest depth (centimeters)		
Years				Years		
6–8	9–11	12–14		6–8	9–11	12–14
−1.0	−1.3	−1.0	16.1–16.5	−0.8	−1.0	−0.6
−0.9	−1.2	−0.8	16.6–17.0	−0.7	−0.8	−0.4
−0.8	−1.0	−0.6	17.1–17.5	−0.6	−0.7	∓0.2
−0.7	−0.8	−0.4	17.6–18.0	−0.6	−0.5	−0.0
−0.6	−0.7	∓0.2	18.1–18.5	−0.5	−0.3	+0.2
−0.6	−0.5	−0.0	18.6–19.0	−0.4	∓0.2	+0.4
−0.5	−0.3	+0.2	19.1–19.5	−0.3	−0.0	+0.6
−0.4	∓0.2	+0.4	19.6–19.9	−0.2	+0.2	+0.8
−0.3	−0.0	+0.6	20.0–20.4	∓0.1	+0.3	+1.1
−0.2	+0.2	+0.8	20.5–20.9	−0.0	+0.5	+1.3
∓0.1	+0.3	+1.1	21.0–21.4	+0.1	+0.7	+1.5
−0.0	+0.5	+1.3	21.5–21.9	+0.2	+0.8	+1.7
+0.1	+0.7	+1.5	22.0–22.4	+0.3	+1.0	+1.9
+0.2	+0.8	+1.7	22.5–22.9	+0.4	+1.2	+2.1
+0.3	+1.0	+1.9	23.0–23.4	+0.5	+1.3	+2.3
+0.4	+1.2	+2.1	23.5–23.9	+0.6	+1.5	+2.5
+0.5	+1.3	+2.3	24.0–24.4	+0.7	+1.7	+2.7
+0.6	+1.5	+2.5	24.5–24.9	+0.8	+1.8	+3.0
+0.7	+1.7	+2.7	25.0–25.4	+0.9	+2.0	+3.2
+0.8	+1.8	+3.0	25.5–25.9	+0.9	+2.2	+3.4
+0.9	+2.0	+3.2	26.0–26.4	+1.0	+2.3	+3.6
+0.9	+2.2	+3.4	26.5–26.9	+1.1	+2.5	+3.8
+1.0	+2.3	+3.6	27.0–27.4	+1.2	+2.7	+4.0

Now, from the children selected by clinical examination, 72.5 % were correspondingly selected by the modified index, whereas, on the other hand, only 3.4 % of the children not selected by clinical inspection were still selected by the modified index.

Concerning the various measurements in the groups of the clinically not selected, clinically selected, and A.C.H. selected, it is evident that there are only slight age differences in these groups. These differences, however, are statistically not significant, so that we can say that selected children and children non-selected are of the same age. Neither we can note differences in the chest girths, and hip widths, respectively, which are statistically significant, whereas great disparities can be noted in the arm girths, which are remarkably smaller in the selected children than in the non-selected. Thus it is the arm girth which, in agreement with the statement of Franzen and Palmer, forms the fundamental character on which the index is based. This statement is in opposition to the suggestion of Aykroyd, who found an increased anterior-posterior diameter of the chest to be the basis of the index. Nevertheless, both factors lead to the same conclusion: a child selected as suffering from malnutrition is more likely to show a low, or even negative value of this difference (we call it D) between arm girth and chest depth. This fact is in correspondence with our observation that 52 % of all children selected by clinical examination showed negative values of D, whereas negative values in the group non-selected appear in only 4.3 % of the children. These results confirm the fact that a low value of D tends to occur in the children being selected, and it is obvious that this character, in relation to the hip width, is prominent in connection with the problem of assessing the state of nutrition of children.

When we recall the original A.C.H. chart of Franzen, we see that the lowest hip width measurements given is 20 cm, all cases with smaller measurements being pooled in a single group. This could lead to the conclusion that all children with hip widths smaller than 20 cm, and negative values of D, are to be selected. This, however, is in contrast to our observations, and likewise to those of Aykroyd, who, in a publication in 1938, proposed that negative values should, like positive values, be graded in relation to hip widths below 20 cm, and he obtained the corresponding values by extrapolating the A.C.H. curve. Indeed, our observations led to the result that 34 % of all children investigated showed hip widths of below 20 cm.

When we look at the graph of the A.C.H. curve of Franzen, we can state a relatively steep gradient of the curve which leads to the result that, in this figure, many children with hip widths above 20 cm would be selected by the index, who, in reality, are in a normal state of nutrition. Indeed, it is this fact which leads to the great number of nearly 10 % of the children who, though fairly nourished according to their clinical state, are still selected by the index.

On the other hand, the extrapolation of this curve to values below 20 cm immediately yields considerably negative values. Thus it might be concluded that children with hip widths below 20 cm, and slightly negative values of D, may be considered as fairly nourished, in contrast to their clinical state. This fact has been confirmed by our observations, which, applying the extrapolation of the original A.C.H. chart, showed only 29 % of the children beeing selected both by clinical inspection and by the A.C.H. index.

In contrast to this, the modification of the curve, with the curve gradient beeing more levelled, leads to a corresponding selection of 74 % of the children, thus indicating a far better result.

The index could, of course, be varied in many other ways, and further research would be necessary to determine the true optimal position of the line separating the selected cases from the non-selected group. There is no doubt that a reliable index which helps to assess the state of nutrition of children would be very useful. We are quite aware of the fact that our work is only a step in this direction, but we should be glad if it would incite further investigations on this subject.

GOITRE SURVEY IN THE NORTH OF BELGIAN CONGO

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The thyroid pathology we find most frequently in Belgian Congo is the hypertrophy of the gland, the goitre; rarely there is a repercussion of this hypertrophy on the general condition.

One could say that the goitre, like other organic deviations, is one aspect of a constitutional disease, a reaction of stress, especially in the growing organism.

North of the Congo River is an endemic area, where 80-90 % of children before puberty have this hypertrophy of the thyroid, which we divided in three stages:

Stage I: gland only palpable, not visible.
Stage II: hardly visible goitre, diffuse or nodular.
Stage III: goitre easily visible, nodular or diffuse.

In these endemic areas we find, usually in prepuberty, a nodular form, which in our opinion is characteristic for a well developped, often irreversible, goitre.

It is evident that the low iodine content of food and drinking-water in this endemic area brings about a diminution of the iodine pool; this, in its turn, causes the thyroid gland, the important iodine pump, to hypertrophy in order to produce enough thyroid-hormon.

In the north of Belgian Congo, on the right bank of the Congo river, is a great endemic area. Strikingly low is the number of goitres found on the left bank of the river, this side being of alluvial origin, where the right side is of eolein origin.

The tribes living immediately near the water make an exception on this finding: the endemicity starts abruptly among the population of the forest at some distance of the right bank.

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But iodine-deprivation is not the cause of all goitres; always the condition is found more in females than in the male, but in a varying degree in the different areas. For instance: in a tribe with 6 % goitres, 80 % were found in women and 20 % in men; in a series of 220 goitre operations, 67 % were done on women and 33 % on men. This higher incidence in the female could partly be explained by the goitre-uterus fibroma-syndrome, a condition we found in 7 % of the female goitre-patients in Congo. In this syndrome the thyroid condition is often secondary to a hypersecretion of follicle-hormon; we did find it even in regions without any goitre endemicity.

Outside the endemic area we examined many schoolchildren in villages near the river, during investigation about hepato-splenomegaly. These tribes consume fish regularly, which excludes iodine deprivation. Here we found a low incidence of thyroid hypertrophy. Examination of 386 boys, aged 7-16 years, revealed only 3,86 % of hardly palpable hypertrophy of the gland. In 29 % of these children we found hepato-splenomegaly. What is more: of the 13 patients with thyroid hypertrophy, 11 also had a hepatosplenomegaly. This high number of coincidence is tempting to attribute the same pathogenesis to the hypertrophy of the thyroid and the hepatosplenomegaly.

Stanbury believes the ultimate cause to be iodine deficiency; where this is not proven for the tribes we have examined, we must have an open mind for other causes than iodine-deficiency for the formation of sporadic goitres.

Goitres are found in the entire Belgian Congo, with an average of 3 % in prepuberty. This sporadic goitre is physiological and, being as frequent in Europe as in Belgian Congo, does not point to an endemicity. An endemic region is characterized by a higher incidence of goitres, among all the age groups, by the appearance of nodular goitres in prepuberty and, in Belgian Congo, by degenerative phenomena (cretinoidisme and more serious forms of cretinism).

To determine the extent and the intensity of the endemia and the degree of the degenerative phenomena, we have done functional and chemical tests, pathological and autoradiographic studies, and therapeutical tests (surgical or medical).

So far, systematic prophylaxis was not done in the examined area.

SURVEY

After many years of thorough investigation, we have determined the extension of the region of goitre-endemicity. On the eastern side it is sharply limited by the mountains and in the south by the Congo river. The transition to the area of hyperendemicity is also well defined; this starts abruptly around Dungu, which is the Azande territory.

The degree of endemicity varies following the progress of civilisation. Near the missionary and commercial settlements, the endemia becomes less striking. The more one moves away from these centres of variety of food, the higher the endemicity.

To a certain degree, not yet determined, inbreeding favorises cretinism to become apparent, this being more serious and frequent in villages that are inaccessible, but by long and difficult paths.

We have studied all the manifestations of cretinism, affecting in some areas 25 % and even more of the population, including some rare cases of congenital goitre with hypermegalie (a child of 4,3 kg from a mother of 45 kg).

Among the infants we have found retarded children, not walking at the age of 2 years, with absent, delayed or bad dentition, strabism or aphonia. Very often we have seen a spastic paraplegia of the legs in the growing child, sometimes also affecting the arms, which could be compared to Littles disease, causing pronounced *genu vara* with atrophia of the musculature of the lower extremities. A great deal of these children were also deaf mute.

The adolescent cretinoid shows signs of an incomplete puberty, pubic and axillary hairs are missing, breasts and testicles are underdevelopped. Notwithstanding certain anomalies, these patients may very well be fertile: we have seen pregnancy in a deaf mute cretinoid with generalized hypertonia and advanced anaemia.

The rest of the population in these endemic areas has a goitre and shows sometimes degenerative characteristics like deaf mutity, debility or imbecility. Other investigators have found in these regions a distressing decrease in birth rate. To a certain degree, these degenerative changes can be attributed to inbreeding and can, in so far, be corrected by a new ethnological policy and by the progress of civilisation.

The goitres become nodular, and characteristic is a firm polylobulated hypertrophia of the whole gland. The isthmus has a tendency to become retrosternal in position.

The most frequent complications are hemorrhage and infection of these nodules; malignancy whoever has been observed only sporadically. This confirms the findings of other investigators who have found seldom a thyroid cancer in the hyperendemic areas. From an endocrinological point of view, hypothyroidism is frequent, where hyperthyroidism is the exception in these areas.

THERAPEUTICAL TESTS

Where there are many practical difficulties in prophylaxis with iodated salt in tropical regions, we were forced to treat the cases that came voluntary to our medical centre, situated in the marginal zone of the endemic area.

There was surgical intervention in a series of 220 patients, the indications for surgery being:

Hyperthyroidism	3 %
Compression of the trachea and more rarely of the recurrent nerves	25 %
Trouble caused by infection (strumitis) or intranodular hemorrhage	10 %
Syndrome of goitre-fibroma...	5 %
Dyspnea on exertion, recurrent bronchitis...	25 %
Heart disease	2 %
Various troubles, aesthetical reasons...	30 %

In this last series the treatment with thyroid extract was not done but after a punction-biopsy of the nodule or the diffuse enlarged gland. Only in the case of fetal adenoma, a thyroidectomy was done; but in cases with macrofollicular goitre, when the punction revealed colloid, we did a therapeutical trial, during 4 to 6 weeks, with doses up to 800 mg of thyroid extract a day, often with good success.

During surgical intervention, varying from enucleation, lobectomy or subtotal thyroidectomy, according to the case, we found in 15 % of the cases a retrosternal goitre, responsible for compression of the trachea.

In the case of unilateral goitre, the heterolateral lobe was always explored.

We did find frequently (10 %) a recurrence after surgical intervention in patients who were not followed up and treated with thyroid-extract in the post-operative period. This recurrence arises in the heterolateral lobe, perhaps from an adenome present in a latent state in this lobe. So, without examination of the opposite lobe during operation, we consider intervention in the other lobe as in effective and avoid surgical intervention without strict indication, i.e. tumor or compression, in underdevelopped countries.

A punction-biopsy will guide us in our hormonal treatment or indicate surgical intervention in the case of an adenoma. This benign tumor should be removed because, especially in the child, where the structures are often papilliferous, it has a tendency to become malignant. In the adult it is said to be susceptible for malignant degeneration, though we have never observed this phenomenon ourselves; but hemorrhage or infection in the adenoma are frequent. Sooner or later these complications will call for urgent surgical intervention.

Iodine acts indirectly by the formation of thyroid-hormone. Regular daily doses of 1 mg and more might cause hyperthyroidism in these goitre patients. As a curative we prefer to use thyroid extract or thyroxine and as a prevention the smallest possible dose of iodine, which cannot cause an iodine-Basedow (100 μg a day).

PATHOLOGY

At birth the thyroid gland has a weight of 1.5 g in babies from euthyroid parents. At 6 months we observe average weights of 2 g, at 2 years 5 g, and at 5 years 10 g. The thyroid of the adult has a weight of 30 g (range 20 g till 40 g), like the European. Mortensen found equal weights, depending on age, undepending on sexe.

The weight of 106 surgically removed goitres varied from 50-1,800 g, with the largest number between 100-200 g. Macroscopically the fetal adenoma can be recognized by the firm consistence, due to the formation in the centre of a white-golden solid tissue, and cysts with clots of organized blood.

The colloidal goitre is red-brown and contains a gelatinous hardened substance. In all these goitres, cysts, with a crusted lining of degenerated often calcified material, are present. These calcifications are present in 13 % of the goitres and are detectable by X-ray.

A goitre that seems clinically diffuse is but a transitional phase towards the nodular form. Indeed, in 23 of these diffuse goitres, we found in 22 patients associated with it a fetal adenoma, or a colloidal goitre, or a microvesicular goitre.

Histological examination of 193 surgically removed goitres:

Revealed, as far as the follicules are concerned:

Fetal adenoma	26 %
Microvesicular goitre	27 %
Macrovesicular colloidal goitre in rest	16 %
Macrovesicular goitre with secondary reaction	23 %
Mixed cases	7 %
Chronic strumitis	0,5 %
Lymphosarcoma of the thyroid	0,5 %

Examination of the connective tissue of 118 removed goitres:

Loose connective tissue	25 %
Sclerosis	20 %
Hemorrhage	19 %
Myxomatous degeneration	11 %
Inflammation	5 %
Fine striae with connective tissue	4 %
Hyalinisation	6 %
Calcification	9 %
Embryonal rests of keratinized Malpighi epithelium	1 %

FUNCTIONAL TESTS

Though we studied the basal metabolism in more than 400 goitre-patients, this test presents too great an error to be a specific measurement for the function of the thyroid-gland, in the complex scale of tropical pathology. Whoever it is useful in the follow-up of one and the same patient after surgery and during thyroidine-therapy. Measurement of the B.M. of a Congolese in complete rest gives normal figures between — 5 % to + 10 %.

Methods based on radioactive isotopes can be used on a large scale and give an idea of the specific function of the gland. Tracer doses of R.I. 131 taken by mouth under standardized conditions showed the avidity of the gland for iodine of goitran patients and of patients operated upon more than two years previously, without postoperative thyroidine treatment.

We studied the uptake and the urinary excretion. These two methods give the same results: the excretion of iodine is inversely proportionate to the uptake of the gland.

We studied also the influence of the thiocyanate -ion, present in manioc, on the elimination of radioactive iodine. The doses of thiocyanate necessary to obtain a blocking effect in the uptake of the gland, and thus to accellerate renal excretion, did in no way correspond with the doses we found after retting of manioc. The problem of goitrogenous substances based on thiocyanate appears to be of no importance in the pathogenesis of the goitres studied by our group.

Normal figures concerning uptake and excretion, as they are given for the civilized countries, can also be used for the population in our area (40 % uptake, after 24 hours, range 2 till 77 %). On the other hand, we must be sure of the fact that the patient does not have a tendency to retain water and chlorides, as in hypoproteinaemia, renal or cardiac disease. The iodine tracer, following to a great extent the space occupied by the chlorides, runs the risk to get lost in the interstitial fluids and to be withdrawn from the thyroid-gland; in this way we have an extra-thyroidal fixation of impredictable duration, which falsifies the results of uptake and output.

CHEMICAL TESTS

The cholesterol-level of the blood is supposed to be elevated in patients with hypothyroidium. In the heart of the endemic area whoever we found a reduced value, that is to say: 134 ± 26 mg/100 cm^3 serum; this test was done in 100 goitran patients, 40 male and 60 female of an average of 35 years of age. For Europeans of the same age group other workers have found values of 175 mg percent.

Three different samples of 100 g of crude salt, as it is consumed by the population of remote villages, contained 200 μg of iodine only; whoever a forth sample contained 3 mg/100 g crude salt. Without any doubt, important factors in this matter are the origine of the salt and the way of stocking. All the analysis done on this subject show the inreliability of crude salt, so far as effective prophylaxis in tropical regions is concerned.

Drinking-water locally supplied contained a low level of iodine. With Prof. De Visscher of the University of Louvain, we examined till now 14 samples of different points of the high endemic region and found an average of 0.213 μg/liter of water. In the marginal area of the hyperendemic region we found 0.381 γ in 6 samples. Further analysis will be done, but, as a matter of fact, we observe that iodine content of water is low.

AUTORADIOGRAPHY OF THE THYROID

To get an idea of the functional modalities of goitre, we have given doses of radioactive iodine up to 1/2 millicurie before operating, and afterwards exposed a slice of the gland to photosensible paper. In this way, we obtained pictures of fetal adenoma, parenchymatous microvesicular goitre, macrovesicular goitre in rest, and especially of cases that were mixed, as far as function of the gland goes. In one case of a physiological goitre post-puberty we found a homogeneous microvesicular structure.

We are sure that it is the disparity of functions that represents the origin of a goitre, whereas the hyperplasia of the hyperfunctioning parts is the result.

CONCLUSIONS

To end this resume we could say that the goitre endemia in the north of Congo represents an area of serious hyperendemicity which, in the actual absence of iodine prophylaxis, diminishes near European infiltration and near points of isolated medical action.

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CAMPANHA CONTRA O BÓCIO ENDÉMICO NO BRASIL

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Com o presente trabalho pretendemos apresentar aos VI Congressos de Medicina Tropical e Malária o que vem fazendo o Ministério da Saúde do Brasil na luta contra o bócio endémico.

Este informe, na verdade, visa, a nosso ver, dois pontos importantes: primeiro, mostrar aos Srs. Delegados como se organizou e vem sendo conduzida uma verdadeira campanha de massa contra o bócio endémico num país de 60 milhões de habitantes a 8 500 000 km² e, segundo, acentuar o emprego do iodato de potássio, em vez do iodeto, como base dessa campanha.

O bócio endémico, simples ou colóide, vulgarmente denominado *papo*, inclui-se entre as doenças de carência — deficiência de iodo na alimentação — e caracteriza-se pelo aumento de volume da tiróide, sem apresentar carácter inflamatório, degenerativo, nem corresponder a certos quistos, como dermóides, hidáticos, etc. O bócio endémico é, na realidade, um tumor benigno do corpo tiróide, sem reacção hiper ou hipofuncional (1), traduzindo-se, segundo D. Marine e outros, numa hipertrofia compensadora da glândula tiróide.

O bócio endémico é, há mais de um século, referido no Brasil por cientistas — A. Saint Hilaire, J. B. Spix, von Martius — que percorreram, principalmente, áreas de Goiás, Minas Gerais e São Paulo.

Durante muito tempo foi o bócio endémico confundido com a doença de Chagas, e só após os excelentes trabalhos de A. Lobo

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Leite ficou perfeitamente demonstrada a inexistência de relação etiológica entre as duas endemias.

Assim, apesar de conhecido e estudado por numerosos pesquisadores, e não obstante os trabalhos de David Marine e O. P. Kimball, a partir de 1915 (3) relativos à eficácia do iodo através do sal de cozinha na prevenção do bócio endémico, não houve nenhuma campanha organizada, de âmbito nacional, contra essa endemia.

Com a deflagração da segunda guerra mundial, trazendo a primeiro plano o homem como factor primordial e insubstituível, lembrou-se então o poder público de que milhões de brasileiros atingidos pelo bócio endémico não podiam ficar à margem da nossa vida militar e económica.

Surge daí a primeira medida oficial, sugerida em 1944 pelo Prof. Josué de Castro, então director do Serviço Técnico da Alimentação Nacional, e traduzida em portaria do coordenador da Mobilização Económica, instituindo a obrigatoriedade da iodação do sal de consumo destinado às zonas bocígenas do País (4).

Essa medida, infelizmente, não chegou a ser posta em prática, pois o sucessor do então coordenador da Mobilização Económica tornou sem efeito a referida portaria.

Terminada a guerra, reconstitucionalizado o País, cuidou-se então de medidas legais visando a luta contra o bócio endémico: a apresentação do Projecto n.º 957/48, que tramitou morosamente durante cinco anos e só em 1953 se transformou na Lei n.º 1944, de 14 de Agosto de 1953. Esta lei é o instrumento legal determinante da obrigatoriedade da iodação do sal destinado a consumo alimentar nas áreas bocígenas do País, após o seu levantamento pelo Ministério da Saúde.

A tarefa de levantamento das áreas bocígenas no Brasil foi magnìficamente executada pela Divisão de Organização Sanitária, do Ministério da Saúde, cujo relatório: *Áreas Bocígenas do Brasil* (6), permitiu o conhecimento da incidência dessa endemia em todo País e o consequente esquema de trabalhos futuros.

Para se ter uma ideia nítida da extensão dessa endemia, de acordo com o citado relatório, basta examinar-se o quadro 1.

Verificou-se, assim, que o bócio endémico existe, pràticamente, em todo território nacional, embora, face à Lei n.º 1944, já citada, não se possa considerá-lo endémico nos estados de Amazonas, Pará, Piauí, Baía, Paraíba, Ceará, Rio Grande do Norte, Alagoas, Pernambuco e Sergipe e nos territórios do Rio Branco e Amapá.

QUADRO 1 — *Incidência de bócio por sexo e por município segundo as unidades da Federação*

Unidades da Federação	Número de municípios		Incidência média (por cento)		
	Existentes	Pesquisados	M	F	Ambos os sexos
Guaporé	2	1	16,1	20,6	17,1
Acre	7	1	33,9	53,8	46,0
Rio Branco	1	1	—	9,6	—
Amapá	4	2	0,3	2,4	1,4
Pará	59	20	5,2	8,6	7,2
Maranhão	87	—	—	—	—
Pernambuco	102	52	0,01	0,02	0,02
Ceará	96	40	0,4	0,6	0,5
Paraíba	54	33	0,8	1,1	1,0
Piauí	62	14	2,9	7,4	5,5
Rio Grande do Norte	—	—	—	—	—
Alagoas	41	12	—	0,06	0,03
Baía	170	67	—	—	—
Sergipe	61	10	—	0,04	0,02
Minas Gerais	485	225	32,5	36,8	34,6
Espírito Santo	37	18	18,9	26,7	21,51
Distrito Federal	1	1	17,0	25,1	21,4
Rio de Janeiro	59	43	25,7	32,3	28,8
São Paulo	435	366	27,3	30,0	28,5
Paraná	149	70	24,5	27,2	24,0
Santa Catarina	67	39	24,1	33,6	28,5
Rio Grande do Sul	112	67	18,4	20,8	19,5
Mato Grosso	59	15	54,2	63,5	58,9
Goiás	126	8	28,2	40,1	34,0
Amazonas	25	11	6,6	11,5	9,2

Estes estados e territórios, note-se bem, não estão isentos de bócio simples, embora apresentem índices inferiores a 15 %, o que justifica plenamente a generalização do uso do sal iodado, a fim de evitar o aumento de sua incidência.

Convém esclarecer ainda que em diversas unidades da Federação, acima citadas, o bócio simples se apresenta com carácter endémico em algumas zonas fisiográficas das referidas unidades.

Aliás, cabe aqui mencionar-se o excelente trabalho do tenente-coronel médico Dr. N. S. Mitke, baseado em estatística elaborada pela Directoria de Saúde do Exército, no qual o autor, revendo as 436 190 inspecções de saúde referentes às classes de convocados de 1929, 1930 e 1931, chama a atenção para o problema do bócio endémico, que se apresentava como a terceira causa de incapacidade para o serviço militar, numa percentagem de 3,07 % para todo o País e de 11 % para a Região Militar Sul (5).

É esse trabalho, a nosso ver, o mais completo estudo sobre a incidência do bócio simples no Brasil, antes do relatório da Divisão de Organização Sanitária a que nos referimos anteriormente.

Ambos os estudos se completam e permitem uma justa estimativa do número de casos ora existentes no País — cerca de 12 000 000 —, cuja distribuição por regiões fisiográficas é dada no quadro 2.

Antes de abordarmos o problema da profilaxia do bócio endémico é interessante apresentarmos alguns dados epidemiológicos, obtidos nos vários inquéritos, sobre a distribuição dessa endemia no Brasil.

Coincidindo com investigações feitas nos demais países, os inquéritos realizados entre nós revelaram uma maior predominância do bócio no sexo feminino, sobretudo nas áreas de prevalência inferior a 30 %.

Quanto à incidência por idade, o nosso companheiro de Coordenação, Dr. Fausto Magalhães Silveira, orientou seus inquéritos, especialmente no estado do Maranhão e Triângulo Mineiro, dividindo os pacientes examinados em dois grupos etários: 7 a 11 e 12 a 18 anos. Os resultados apurados revelam uma incidência 9 % maior no segundo grupo etário, reflectindo certamente uma maior exigência do organismo na fase da puberdade.

Referentemente à cor, o grau de incidência entre brancos, pardos e pretos é pràticamente nulo.

Finalmente, outro dado importante a mencionar nos inquéritos realizados no Brasil é o referente a altitude. O relatório da Divisão de Organização Sanitária, já referido, conclui pela inexistência de correlação entre bócio endémico e altitude.

QUADRO 2 — *Prevalência do bócio, segundo as regiões fisiográficas do Brasil, e estimativa do número de casos por região*

Regiões fisiográficas	Estados e territórios	População	Prevalência (por cento)	Estimativa de casos de bócio
Centro-oeste	Mato Grosso Goiás	2 200 000	53,8	1 150 000
Sul	São Paulo Paraná Santa Catarina Rio Grande do Sul	20 000 000	27,7	5 500 000
Leste meridional	Minas Gerais Espírito Santo Rio de Janeiro Distrito Federal	16 000 000	27,0	4 300 000
Nordeste ocidental	Maranhão Piauí	3 100 000	11,8	350 000
Norte	Amazonas Pará Acre Amapá Rio Branco Rondónia	2 300 000	9,4	210 000
Nordeste oriental	Ceará Rio Grande do Norte Paraíba Pernambuco Alagoas	12 000 000	0,6	70 000

Criado o Departamento Nacional de Endemias Rurais e nele instituída a Coordenação da Campanha contra o bócio endêmico, em Abril de 1956, providenciou-se imediatamente o planejamento e a execução da luta contra essa endemia à base de quatro *étapes* fundamentais:

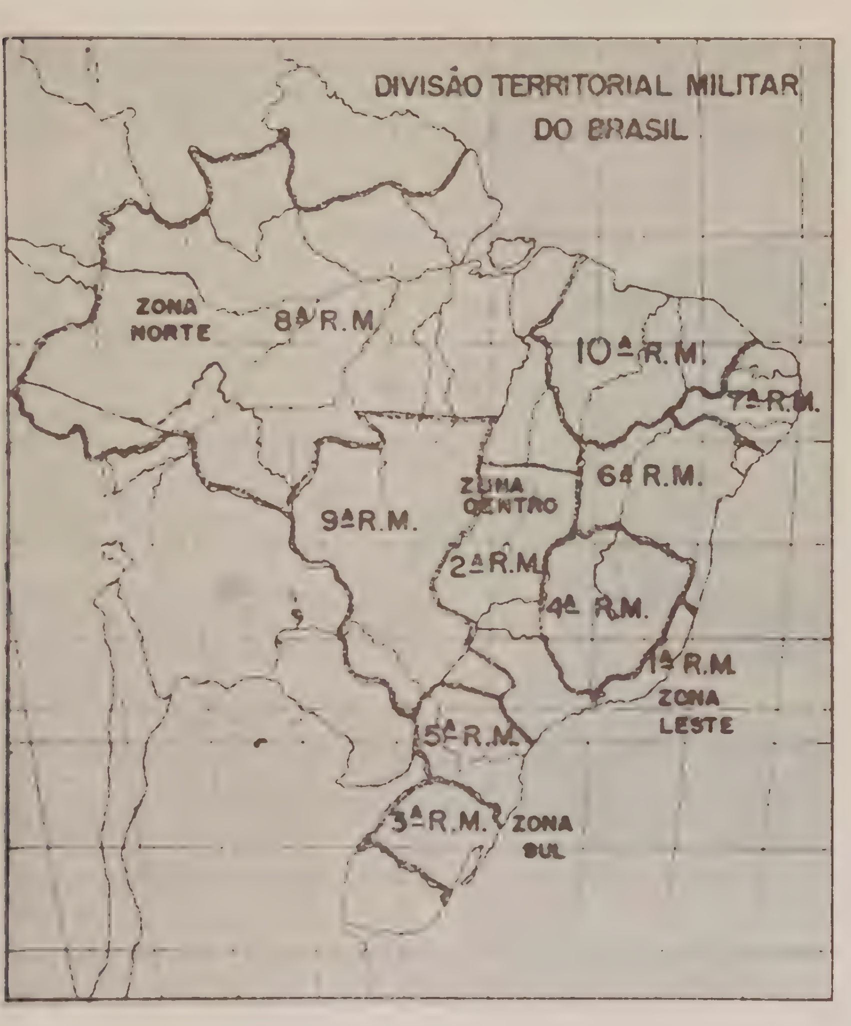

DIVISÃO TERRITORIAL MILITAR DO BRASIL
ZONA NORTE
8ª R.M.
10ª R.M.
7ª R.M.
6ª R.M.
ZONA CENTRO
9ª R.M.
2ª R.M.
4ª R.M.
1ª R.M.
ZONA LESTE
5ª R.M.
3ª R.M.
ZONA SUL

Fig. 1 — Divisão territorial militar do Brasil

a) Legislação complementar indispensável à obrigatoriedade da iodação de todo sal destinado a consumo alimentar e maneira de executá-la;

b) Iodação do sal nas refinarias e moinhos, seu *contrôle* sistemático, em colaboração com o Instituto Nacional do Sal e repartições estaduais de saúde;

c) Prosseguimento de inquéritos sobre o bócio endémico nos municípios ainda não trabalhados e estudos e pesquisas sobre o teor de iodo nos alimentos habitualmente consumidos e suas possíveis modificações face à natureza de vasilhame e à cocção; análises das águas de abastecimento de colectividades, não só para determinação do teor de iodo, como também para verificação de seu grau de dureza (bicarbonatos, sulfatos e cloretos, de cálcio ou magnésio), e no solo de áreas destinadas à cultura; estudo, em colaboração com órgãos e associações especializados sobre a fisiopatologia da tiróide; pesquisas relativas ao metabolismo do iodo (inclusive com iodo radioactivo), a fim de se determinar a melhor quantidade a ser empregada;

d) Inquéritos periódicos, extensos, visando aferir os resultados da iodação do sal no quadro geral da endemia.

Para consecução do previsto na alínea *a*) entrou a Coordenação em entendimentos com o Instituto Nacional do Sal, que promoveu sucessivas reuniões conjuntas, em sua sede, com produtores e distribuidores de sal, a fim de ser redigido ante-decreto fixando as normas a serem adoptadas na iodação do sal no Brasil.

Dessas reuniões resultou o Decreto n.º 39 814, de 17 de Agosto de 1956 (7), publicado no *Diário Oficial* de 20 do referido mês, estabelecendo a obrigatoriedade da iodação do sal moído e refinado para uso alimentar, gradativamente, nas diversas áreas do território nacional.

Amparada, assim, por legislação adequada, a Coordenação cuidou das primeiras providências relativas à obtenção de matéria-prima: iodato de potássio. Como, infelizmente, não havia no mercado nacional esse produto, foi adquirido iodeto de potássio e, no Instituto de Endemias Rurais, transformado em iodato.

Embora a Lei n.º 1944 se refira expressamente a iodeto de potássio, preferiu a Coordenação empregar o iodato, atendendo às seguintes razões: a profilaxia antibocígena pelo iodeto de potássio encontra dificuldades em países de clima quente e húmido, devido à perda de iodo ocasionada pela acção da luz, do calor e das impurezas contidas no próprio sal.

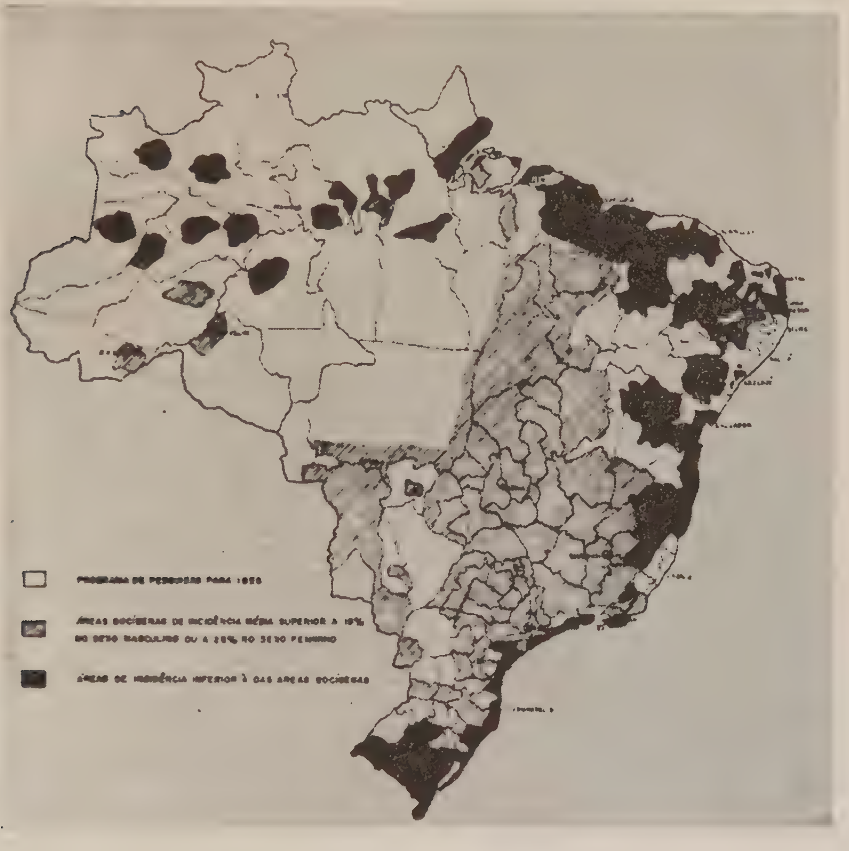

☐ PROGRAMA DE PESQUISAS PARA 1959
▨ ÁREAS BOCÍGENAS DE INCIDÊNCIA MÉDIA SUPERIOR A 10% NO SEXO MASCULINO OU A 20% NO SEXO FEMININO
■ ÁREAS DE INCIDÊNCIA INFERIOR À DAS ÁREAS BOCÍGENAS

Fig. 2 — Incidência do bócio endémico

Experiências realizadas no México em 1952 provaram que o iodo adicionado ao sal é mais estável sob a forma de iodato do que sob a de iodeto. Doutra parte, segundo experiências de laboratório efectuadas na Inglaterra e na Índia, sabe-se que, igualmente sob a forma de iodato, o iodo é mobilizado pela tiróide, não exercendo nenhum efeito tóxico sobre o organismo (8).

Para maior segurança do êxito da campanha procedeu-se, em 6 de Abril de 1956, nas Usinas Perynas, em Cabo Frio, estado do Rio de Janeiro, à iodação experimental de 600 kg de sal refinado, retirando-se desse total amostras de 30 kg (em sacos de aniagem), que foram remetidas para sedes de circunscrições de São Paulo, Goiás, Mato Grosso e Minas Gerais e, dessas cidades, devolvidas ao Instituto de Endemias, a fim de serem examinadas para verificação do teor de iodo, face às condições gerais do ambiente, tais como humidade, calor, luminosidade e outras.

Desse estudo experimental resultaram as seguintes conclusões, apresentadas por Ernest Paulin e José Pedro Pereira, do Instituto Nacional de Endemias Rurais:

1) O sal iodatado, quando armazenado em sacos de polietileno, não perdeu quantidade apreciável de iodato, durante 10 meses, em temperatura ambiente, mas a distribuição da droga no sal não se manteve constante durante esse período.

2) O mesmo sal, armazenado em sacos de algodão em vários pontos do País, mostrou variações no seu teor de iodato. Diferentes valores foram obtidos tanto nas análises das amostras tiradas do mesmo saco como entre os sacos da mesma partida. A menor diferença em relação ao valor inicial foi observada no lugar de menor humidade relativa e a maior diferença no lugar de maior humidade relativa.

3) Quando o sal iodatado foi aquecido em estufa, a perda de iodato foi proporcional à temperatura e às impurezas do sal. Um aquecimento prévio do sal a 450°C destruiu as impurezas que produziram a decomposição do iodato.

4) A sensibilidade do iodato à temperatura elevada, em mistura com o sal de cozinha, já se faz sentir em 105°C, onde normalmente se seca o sal antes de ser analisado. Análises paralelas mostraram uma perda de 18 %, em média, nas amostras submetidas à secagem.

5) O iodato de potássio permanece quìmicamente inalterado durante o armazenamento cerca de um ano, em temperatura ambiente.

6) A distribuição do iodato de potássio no sal durante o armazenamento não permanece uniforme. O grau de desvio da uniformi-

dade parece ser proporcional à humidade relativa do lugar onde o sal foi armazenado.

7) Há uma tendência pela qual a concentração do iodato é maior no centro da massa de sal e é menor nas camadas periféricas.

8) O sal de cozinha, não refinado, tem sempre impurezas orgânicas que decompõem o iodato em temperatura elevada, isto é, acima de 100°C.

9) O aquecimento do sal a 450°C, antes de ser misturado com iodato, destrói todas as impurezas que possam, mais tarde, reduzir o iodato.

10) Para determinar o teor de iodo no sal não se deve fazer a secagem prévia, pois o aquecimento a 105°C reduz o resultado analítico (10).

Paralelamente às medidas acima, a Coordenação reestudou os numerosos dados recebidos da Divisão de Organização Sanitária e cuidou de completar o levantamento dos índices bocígenos, sobretudo em áreas de maior importância, tais como o estado do Maranhão (sobre o qual não havia nenhum levantamento sistematizado) e a zona do Triângulo Mineiro. Esse trabalho foi cuidadosamente realizado, no período de 23 de Setembro a 29 de Novembro de 1956, pelo Dr. Fausto Magalhães, médico sanitarista deste Departamento. Os índices encontrados foram, para o estado do Maranhão, sensìvelmente altos em toda a área compreendida abaixo do paralelo 5, com índice superior a 20 %, e, quanto ao Triângulo Mineiro, o levantamento revelou índices acima de 55 %.

Quanto à alínea *b*) — iodação do sal nas refinarias e moagens —, a Coordenação, inicialmente, procurou conhecer, através do Instituto Nacional do Sal, a produção salineira do País e sua localização.

Essa apresenta-se de acordo com o quadro 3.

O quadro 3 mostra que, apesar de 80 % da produção de sal se encontrar em três estados — Rio Grande do Norte, 60 %, Rio de Janeiro, 15,2 %, e Ceará, 12,6 % —, o beneficiamento se faz, sobretudo, nas áreas de maior consumo: São Paulo, Distrito Federal, estado do Rio e Rio Grande do Sul.

Embora existam, de acordo com o Instituto Nacional do Sal, 892 salinas em todo território nacional, na realidade a quase totalidade da moagem e refinação está concentrada em dez grandes indústrias, facilitando destarte a tarefa de iodação do sal.

QUADRO 3 — *Distribuição das salinas por unidade da Federação e respectiva produção*

Estados	Número de salinas	Produção	
		Em toneladas	Percentagem
Pará	2	70	0,01
Maranhão	200	22 750	3,25
Piauí	17	15 260	2,18
Ceará	71	88 130	12,59
Rio Grande do Norte	95	416 920	59,56
Paraíba	6	1 680	0,24
Pernambuco	62	2 450	0,35
Alagoas	8	210	0,03
Sergipe	308	36 890	5,27
Baía	15	9 590	1,37
Rio de Janeiro	108	106 050	15,15
	892	700 000	100,00

A iodação do sal vem-se fazendo de acordo com o quadro abaixo:

QUADRO 4 — *Produção de sal iodado por unidade da Federação*

Unidade da Federação	Produção anual (toneladas
Distrito Federal	61 000
Estado do Rio de Janeiro	114 600
Estado de São Paulo	150 000
Rio Grande do Sul	51 500
Paraná	28 000
Total anual	405 100

A produção constante do quadro 4 destina-se ainda a outras unidades da Federação, que não possuem moagens próprias, ou as possuem insuficientes para suas necessidades, distribuindo-se de acordo com o quadro 5.

Os dados apresentados são expressivos e mostram que 65 % da população do País já está beneficiando do uso do sal iodado.

QUADRO 5 — *Relação dos estados consumidores de sal iodado e população beneficiada*

Estados e Distrito Federal	Área (quilômetros quadrados)	População
Estado de Minas Gerais.	581 975	9 261 350
Estado de São Paulo	274 222	10 961 308
Estado do Paraná	200 300	2 538 656
Estado de Santa Catarina	94 280	1 872 603
Estado do Rio Grande do Sul . . .	267 456	4 997 785
Estado de Goiás	622 555	1 457 905
Estado de Mato Grosso.	1 254 821	626 452
Estado do Espírito Santo	39 577	1 033 875
Estado do Rio de Janeiro	41 666	2 756 633
Distrito Federal	1 171	2 852 941
Estado da Baía (parte)	200 000	2 200 000
Mantena (região contestada). . . .	10 137	200 000
Total	3 561 160	40 759 508

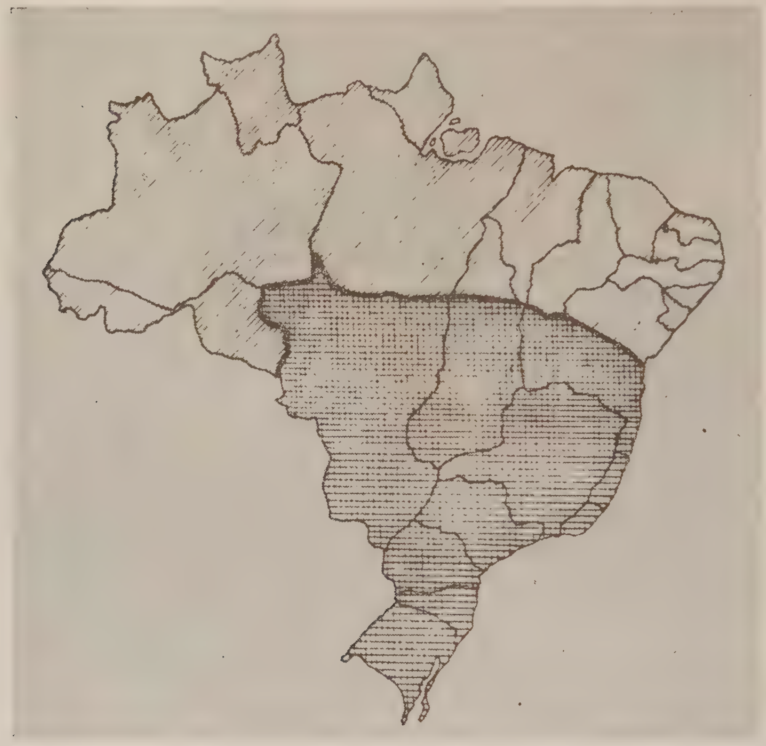
Revelam ainda os mesmos dados que 82 % de todo sal beneficiado (refinado e moído) já vem sendo distribuído às populações devidamente iodado.

A Coordenação concordou em permitir que as indústrias usassem iodato de potássio até 50 % acima da quota estabelecida em lei, tendo em vista as mesmas razões que levaram as autoridades sanitárias suíças a igual procedimento.

Essa tolerância decorreu da apreciação das primeiras centenas de resultados de análises do teor do iodo na produção examinada.

De acordo com a legislação, deveriam ser empregados 0,010 g de iodo metalóidico para cada 1000 g de cloreto de sódio, ou 0,017 g de iodato de potássio, que é o seu equivalente.

No estudo das causas determinantes das variações do teor de iodo encontrado nas análises verificou-se que essa flutuação era devida a dois factores fundamentais: variações da humidade relativa no ambiente e desgaste de peças essenciais à moagem e à trituração do



▨ Área ainda a ser beneficiada com sal iodado em 1958

▦ Área coberta pelo sal iodado, representando:

65 % da população .... 42 000 000 hab.
41 % da superfície do Brasil ...... 3 500 000 km²

Fig. 3 — Penetração do sal iodado nas diversas unidades da Federação (1958)

sal, desgaste este perturbador da vazão de sal, moído ou triturado, por unidade de tempo.

Para remover esse óbice e manter o sal beneficiado dentro dos limites necessários à profilaxia do bócio endémico permitiu-se um acréscimo de até 50 % do estabelecido em lei.

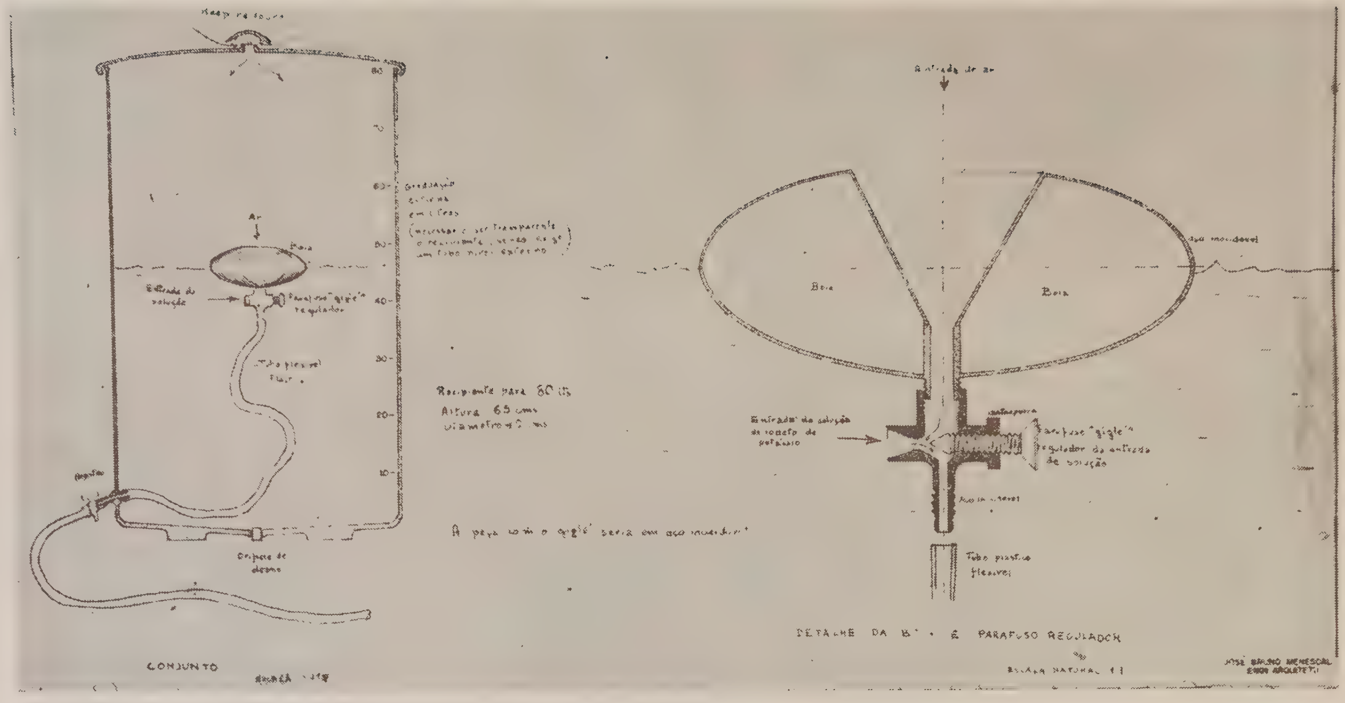

80
60-
50-
40-
30-
20-
10-
Ar
Boia
Entrada da solução
Parafuso "gigle" regulador
Tubo flexível
Recipiente para 80 lts
Altura 65 cms
A peça com o gigle seria em aço inoxidável
CONJUNTO
Entrada de ar
Boia
Boia
Entrada da solução de iodato de potássio
contraporca
Parafuso "gigle" regulador da entrada de solução
Tubo plastico flexível
DETALHE DA BÓIA E PARAFUSO REGULADOR
ESCALA NATURAL 1:1
JOSÉ BRUNO MENESCAL
ENG. ARQUITETO

Fig. 4 — Tipo de dosador de iodato de potássio projectado pelo Departamento Nacional de Endemias Rurais para iodação de sal

Essa tolerância vem-se revelando necessária, e as análises dos últimos meses, efectuadas na produção de beneficiamento de sal, no Distrito Federal, estados do Rio e de São Paulo, mostram que o teor encontrado oscila dentro dos limites recomendados.

Cabe ressaltar, nesta alínea, que a Coordenação se fixou pelo processo húmido à base de solução titulada de iodato de potássio a 2 % (cada mililitro corresponde a 0,017 g de iodato), por ser de uso fácil e permitir maior homogenização durante o beneficiamento, com aumento desprezível de humidade (1 %).

A Coordenação vem recomendando equipamento de acordo com o modelo constante do anexo.

Quanto à alínea *c*) — estudos e pesquisas —, o programa em desenvolvimento vem-se realizando em colaboração com o Instituto de Nutrição, com o qual o Departamento Nacional de Endemias Rurais celebrou um acordo a fim de, no referido Instituto, serem feitas as necessárias pesquisas relativas ao teor do iodo nos alimentos habitualmente consumidos nas diversas regiões do Brasil, nas águas de abastecimento das colectividades e no solo de áreas destinadas à cultura, além de outros problemas que interessam ao perfeito conhecimento do bócio endémico.

Finalmente, quanto à alínea *d*), programou a Coordenação a realização de inquéritos de *contrôle* para a avaliação do êxito da medida profiláctica adoptada.

Trienalmente serão, então, feitos amplos inquéritos abrangendo o País.

RESUMO

O bócio endémico constitui uma das grandes endemias no Brasil, estimando-se em cerca de 12 000 000 o número de portadores de hipertrofia da glândula tiróide.

Os inquéritos realizados mostraram que a maior incidência dessa endemia se encontra nas regiões fisiográficas centro-oeste, sul e leste meridional.

Todas essas regiões fisiográficas revelam uma incidência acima de 25 % entre os escolares de ambos os sexos. Essas regiões fisiográficas abrangem os grandes centros industriais do País.

As regiões nordeste ocidental e norte apresentam uma incidência média de 10 % entre os escolares de ambos os sexos.

Atendendo a que a endemia bocígena é de enorme extensão, cuidou o Departamento Nacional de Endemias Rurais, do Ministério da Saúde, de planejar um

programa de profilaxia, à base de sal iodado, capaz de assegurar a toda população brasileira 100y diárias de iodo metalóidico.

É, no género, a maior campanha que já se fez no mundo para combate organizado ao bócio endémico, à base de iodato de potássio.

Esta Campanha obedece a três fases:

a) Legislação permitindo a obrigatoriedade da iodação do sal destinado a consumo alimentar;

b) Iodação do sal, referido na alínea anterior, por parte dos beneficiadores desse produto;

c) *Contrôle* permanente, por parte da Campanha contra o Bócio Endémico, do sal distribuído ao consumo alimentar, a fim de se verificar o seu teor de iodato;

d) Inquéritos e pesquisas visando o conhecimento da redução dessa endemia em áreas já anteriormente investigadas, bem como o estudo do teor de iodo em águas de abastecimento e em alimentos habitualmente consumidos.

Lei n.º 1944, de 14 de Agosto de 1953

Artigo 1.º Nas áreas bocígenas do País, a venda de sal refinado ou moído, para consumo alimentar, só será permitida quando devidamente iodetado, excluído o sal destinado à indústria e à pecuária.

Art. 2.º Para os efeitos desta lei, compreende-se por iodetação a adição de iodo na proporção de 10 mg de cloreto de sódio, mediante quantidades equivalentes e íntima mistura com um dos seus compostos: iodeto de sódio ou iodeto de potássio.

Art. 3.º O Ministério da Educação e Saúde, por intermédio de seus órgãos especializados, em articulação com os departamentos de saúde dos estados interessados, providenciará, até cento e oitenta dias após a publicação desta lei, a delimitação das áreas bocígenas do País.

§ 1.º O grau de endemicidade será determinado mediante percentagem de positividade de casos com hipertrofia glandular tiroidiana, entre crianças em idade escolar de ambos os sexos, considerados separadamente.

§ 2.º Reputar-se-ão áreas bocígenas, para os efeitos desta lei, as localidades onde o índice endémico for superior a 15 % (quinze por cento) para as crianças do sexo masculino e 25 % (vinte e cinco por cento) para as do sexo feminino.

Art. 4.º O despacho do sal refinado ou moído, para consumo alimentar, destinado às áreas bocígenas do País, a partir da vigência desta lei, sòmente será permitido depois de submetido o produto ao processo de iodetação e uma vez inscritas as palavras «sal iodetado» nas sacarias ou invólucros.

§ único. O sal de que trata este artigo enquadrar-se-á na menor tarifa ferroviária adoptada para o cloreto de sódio nas estradas de ferro do País.

Art. 5.º O Governo Federal, por intermédio do Ministério da Educação e Saúde, providenciará para que o Instituto Nacional do Sal organize nos estados do Rio de Janeiro e do Rio Grande do Norte, dentro de seis meses da data da publicação desta lei, uma instalação especializada para a iodetação do sal refinado ou moído, destinado às áreas bocígenas do País, a ser distribuído pelos comerciantes de sal.

§ único. É permitido a qualquer salineiro ou distribuidor de sal instalar usina própria para a iodetação do sal, devendo o Instituto Nacional do Sal, em colaboração com os órgãos a que se refere o artigo 3.º desta lei, prestar-lhe assistência técnica.

Art. 6.º Será permitido, para a venda de sal iodetado, um acréscimo até o máximo de Cr$ 0,40 (quarenta centavos), por quilo, sobre os preços vigentes, na ocasião, para o sal comum.

Art. 7.º É assegurado, através do Instituto Nacional do Sal, ou de particulares que se proponham a fazer a iodetação do produto, o abastecimento das zonas bocígenas, nas quais não será permitido o comércio de sal iodetado, sob pena de apreensão e multa equivalente a duas vezes o seu valor, no caso de reincidência.

§ único. Se não for possível a apreensão do sal, por já ter sido dado ao consumo, o infractor pagará ao Instituto Nacional do Sal, para os fins estabelecidos no artigo 41.º, § 2.º, do Decreto-Lei n.º 2398, de 11 de Julho de 1940, importância correspondente ao duplo do valor do produto irregularmente vendido.

Art. 8.º O processo, para execução das penalidades estabelecidas na presente lei, será feito na forma do disposto no artigo 42.º, §§ 1.º, 2.º e 3.º, do Decreto-Lei n.º 2398, de 11 de Julho de 1940.

Art. 9.º O Governo Federal, por intermédio do Ministério da Educação e Saúde, do Instituto Nacional do Sal e de todos os meios de divulgação do que dispõe, fará nas zonas bocígenas do País ampla propaganda dos benefícios do uso do sal iodetado na profilaxia e combate do bócio endémico.

Art. 10.º Esta lei entrará em vigor sessenta dias após a data da sua publicação.

Art. 11.º Revogam-se as disposições em contrário.

Senado Federal, 14 de Agosto de 1953. — João Café Filho. — *Diário Oficial* de 20 de Agosto de 1953.

Decreto n.º 39 814, de 17 de Agosto de 1956

Delimita as áreas bocígenas do Brasil, dispõe sobre o uso do sal iodado e dá outras providências

O Presidente da República, usando da atribuição que lhe confere o artigo 87.º, n.º 1.º, da Constituição, e nos termos da Lei n.º 1944, de 14 de Agosto de 1953, decreta:

Artigo 1.º Para os efeitos da Lei n.º 1944, de 14 de Agosto de 1953, os estados de Mato Grosso, Minas Gerais, Goiás, São Paulo, Rio de Janeiro, Santa Catarina, Paraná, Espírito Santo, Rio Grande do Sul e Baía, bem como o Distrito Federal e os territórios do Acre e do Guaporé, são considerados áreas bocígenas do País, na conformidade do disposto no artigo 3.º da citada lei e segundo os dados levantados pela Divisão de Organização Sanitária do Departamento Nacional de Saúde. do Ministério da Saúde.

Art. 2.º A partir de 1.º de Janeiro de 1957, todas as refinarias ou moinhos de sal dos estados de São Paulo e do Rio de Janeiro e do Distrito Federal só poderão entregar a consumo sal refinado ou moído quando devidamente iodado, na proporção de 10 (dez) miligramas de iodo metálico por quilograma de sal.

Art. 3.º A partir de 1.º de Julho de 1957 a mesma obrigatoriedade do artigo anterior se estenderá a todas as refinarias e moinhos situados nos estados de Mato Grosso, Minas Gerais, Goiás, Santa Catarina, Paraná, Espírito Santo e Rio Grande do Sul.

Art. 4.º A partir de 1.º de Janeiro de 1958 a iodação do sal será obrigatória para o resto do território nacional em que forem assinalados casos de bócio endémico.

Art. 5.º Antes dos prazos previstos nos artigos 2.º, 3.º e 4.º do presente decreto, os estabelecimentos industriais que desejarem iniciar a iodação do sal poderão fazê-lo, desde que comuniquem essa decisão ao Instituto Nacional do Sal e ao Departamento Nacional de Endemias Rurais, do Ministério da Saúde, com minuciosa descrição do processo adoptado e respectivo esquema da maquinaria empregada.

Art. 6.º O Ministério da Saúde, através do Departamento Nacional de Endemias Rurais, e o Instituto Nacional do Sal, com intervalos não superiores a quatro meses, fiscalizarão todos os estabelecimentos que preparam sal iodado, colhendo amostras *in loco* para as devidas análises.

Art. 7.º A embalagem do sal contendo iodo deverá trazer, em lugar visível, os dizeres «Sal Iodado», a fim de se enquadrar na menor tarifa ferroviária, de acordo com o § único do artigo 4.º da Lei n.º 1944, de 14 de Agosto de 1953.

Art. 8.º O Ministério da Saúde providenciará a importação do iodeto de potássio ou sódio necessário à iodação do sal, fornecendo-o aos interessados pelo preço do custo.

Art. 9.º Para o sal moído iodado será permitido um acréscimo de Cr$ 0,40 (quarenta centavos) por quilo sobre os preços vigentes na ocasião para o sal comum.

Art. 10.º O Instituto Nacional do Sal, em articulação com o Departamento Nacional de Endemias Rurais, fará, nas zonas bocígenas do País, ampla propaganda dos benefícios do uso do sal iodado na profilaxia e combate ao bócio endémico.

Art. 11.º Na vigência dos artigos 2.º, 3.º e 4.º deste decreto não será permitido o comércio do sal moído ou refinado não iodado, sob pena de apreensão e multa equivalente ao respectivo valor, dobrada essa multa em caso de reincidência.

§ único. Se não for possível a apreensão do sal, por já ter sido dado ao consumo, o infractor pagará ao Instituto Nacional do Sal, para os fins estabelecidos no artigo 41.º, § 2.º, do Decreto-Lei n.º 2398, de 11 de Julho de 1940, importância correspondente ao duplo valor do produto irregularmente vendido.

Art. 12.º O processo para execução das penalidades estabelecidas no presente decreto será feito na forma do disposto no artigo 4.º, §§ 1.º, 2.º e 3.º, do Decreto-Lei n.º 2398, de 11 de Julho de 1940.

Art. 13.º O presente decreto entrará em vigor na data de sua publicação, revogadas as disposições em contrário.

Rio de Janeiro, 17 de Agosto de 1956; 135.º da Independência e 68.º da República. — Juscelino Kubitschek — *Ernesto Dornelles* — *Maurício de Medeiros.*

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LES HÉPATOSPLÉNOMÉGALIES DE L'ADULTE AFRICAIN À DAKAR

M. PAYET
et
M. ARMENGAUD

L'étude des hépatosplénomégalies de l'Africain adulte pouvait être envisagée comme celle des états pré-cirrhotiques et nous espérions, ce faisant, obtenir des renseignements précis sur l'étiologie des cirrhoses. Nous verrons au cours de cet exposé que nous rencontrons en fait le même problème, celui de la maladie hépatosplénique de l'Africain.

Précisons tout d'abord le terme d'hépatosplénomégalie; il vaudrait mieux le remplacer par celui d'hépatosplénopathie, car si la splénomégalie est constante et forme en quelque sorte le déterminateur commun à toutes nos observations, l'hépatomégalie est inconstante, bien que le foie soit toujours anatomiquement lésé.

Nous pouvons situer cette étude dans l'ensemble de la pathologie hospitalière que nous avons coutume de rencontrer à Dakar, en donnant les résultats statistiques de 242 autopsies effectuées dans notre service durant le même laps de temps: ces autopsies sont celles d'individus décédés de maladies diverses. On constate que 165 rates pèsent plus de 200 g, 63 rates pèsent entre 100 g et 200 g, 14 rates seulement ont un poids inférieur ou égal à 100 g. D'autres statistiques ont été effectuées, en particulier en Nigéria, qui ont montré que la rate des Africains adultes est plus grosse que celle des Européens de même âge. Les splénomégalies sont donc d'observation courante en Afrique, et il est certain qu'un grand nombre de rates d'adultes sont des rates pathologiques.

Étudier *stricto sensu* les splénomégalies, reviendrait donc à étudier l'ensemble de la population africaine, mais les rates percutables

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ou à peine palpables sont difficiles à étudier en clinique et par les épreuves radiologiques électives. Nous n'avons donc retenu dans notre travail que les observations de malades porteurs de grosses rates qui, selon la nomenclature employée à l'heure actuelle par les auteurs, sont des rates n^os^: 2, 3, 4 et 5:

La rate n° 2 dépasse le gril costal de 1 à 2 travers de doigt.
La rate n° 3 atteint le milieu de la distance côte ombilic.
La rate n° 4 atteint l'ombilic.
La rate n° 5 le dépasse.

Nous avons étudié les malades porteurs de grosses rates qui sont passés dans notre service durant une période de près de deux ans: du 1^er^ janvier 1956 au 1^er^ novembre 1957. Le sort et l'élimination des observations incomplètes ou inutilisables nous ont amené à étudier un chiffre très exact de 100 splénomégalies. Ces observations ont été relevées par une seule équipe d'observateurs dans un seul service, qui fut durant 16 mois celui des salles d'hospitalisation de femmes, et ceci explique la forte proportion de femmes envisagées: 53 femmes pour 47 hommes.

Il s'est agi de *malades africains* de races habituellement rencontrées à Dakar:

26 % de Ouolof.
24 % de Toucouleur.
16 % de Soudanais.
15 % de Peulh.
9 % de Casamançais (Diola, Mandiague).
7 % de Maure.
3 % de divers (1 Dahoméen, 1 Côte d'Ivoirien, 1 Cap Verdien).

Ces malades étaient âgés de 14 à 60 ans et la *répartition des âges* est la suivante:

19 malades étaient âgés de 14 à 20 ans.
34 malades étaient âgés de 20 à 30 ans.
28 malades étaient âgés de 30 à 40 ans.
20 malades étaient âgés au delà de 40 ans.

La majorité de nos observations a trait à des adultes jeunes, mais se rencontrent également aux âges avancés de la vie.

Il nous a été difficile de situer la *date exacte de l'apparition de la splénomégalie,* la notion occidentale du temps n'étant pas celle de l'Africain de la brousse. Nous pouvons toutefois donner les chiffres suivants à titre d'indication pour 94 malades:

15 malades présentaient une grosse rate depuis l'enfance.
5 malades avaient constaté leur splénomégalie depuis 1 mois.
3 depuis 3 mois.
11 depuis 6 mois.
23 depuis 1 an.
27 depuis 2 ans.
10 depuis plus de 3 ans.

En général, les malades ayant vu disparaître leurs splénomégalies de l'enfance vers l'âge de 14 ans, ont vu réapparaître une tumeur de leur hypochondre gauche, vers l'âge moyen de la vie et sont venus à l'hôpital, 1 à 2 ans après cette constatation.

Les plaintes qui ont motivé leur hospitalisation n'ont pas toujours été la splénomégalie, la splénalgie, mais sont diverses.

Nous avons ainsi découvert une grosse rate chez 22 malades qui présentaient une affection intercurrente:

Affection pulmonaire (1 DDB, 1 fibrose pulmonaire, 2 artérites pulmonaires, 2 pneumopathies aiguës).
Affection cardiaque (2) (1 rétrécissement mitral, 1 coronarite).
Affection neurologique (6) (1 Guillain Barré, 1 paralysie des S.P.E., 1 alcoolique, 1 mental, 1 cécité, 1 surdité).
Affection digestive (2 dysenteries).
Ulcère phagédénique (1).
Métro-annexite (1).
Grossesse (4).

Ces associations n'intervenaient en rien dans la formation de la grosse rate et ne présentaient aucun caractère particulier, sinon celui d'amener les malades à consulter.

Les 78 autres malades se plaignaient par contre d'un syndrome en relation avec leur splénomégalie, et nous avons pu noter à l'entrée dans le service les plaintes suivantes:

Mauvais état général, 17 fois.
Douleurs abdominales, 16 fois.
Ascite isolée, 16 fois.
Splénomégalie seule, 15 fois.
Fièvre, 14 fois.
Oedèmes isolés des membres inférieurs, 10 fois.
Ictère, 10 fois.
Hématurie, 6 fois.
Anasarque, 5 fois.
Hématémèse, 4 fois.
Hémorragies diverses (métrorragies, épistaxis), 3 fois.
Troubles digestifs, 3 fois.
Douleur hépatique, 2 fois.
Arthralgies, 2 fois.

chaque malade ayant pu présenter un ou plusieurs symptômes associés.

L'examen clinique physique de la splénomégalie elle-même et des signes d'accompagnement nous ont fourni les renseignements suivants sur l'état de la rate, du foie, du cœur et des ganglions.

La *rate* atteint souvent des proportions importantes et nous avons observé:

19 rates n° 1 et 2.
22 rates n° 3.
13 rates n° 4.
46 rates n° 5.
23 d'entre elles étaient douloureuses spontanément ou à la palpation.

L'épreuve d'Ascoli modifiée a été pratiquée 67 fois, et nous avons relevé 40 rates contractiles et 27 rates non contractiles.

Le *foie* est gros dans 61 % des cas, de taille normale dans 39 cas. Parmi les gros foies, 49 sont de consistance ferme évoquant la fibrose, 12 seulement avaient une consistance normale.

Le *cœur* est ordinairement cliniquement, radiologiquement et électriquement normal. Nous devons mettre à part 2 cardiopathies acquises connues (2 rétrécissements mitraux, 1 coronarite). Chez 15 malades, le cœur était globalement augmenté de volume et présentait tous les caractères des cœurs de myocardie.

Les *ganglions* ne sont ordinairement pas perçus. Cependant chez 16 malades nous avons mis en évidence des adénopathies dans les diverses aires ganglionnaires.

Avant d'entreprendre l'étude des différents symptômes que nous venons d'énumérer, il convient de rapporter le résultat des examens paracliniques que nous avons pratiqués.

Ce bilan avait pour but:

De retrouver une cause essentielle à la splénomégalie et nous avons systématiquement recherché les étiologies suivantes: paludisme, syphilis, bilharziose, ankylostomiase, filariose, trypanosomiase, sicklanémie, tuberculose, hémopathies, infections diverses comme typhoïde, hépatite ictérigène;

D'étudier l'état du foie par les épreuves biologiques habituelles (dosage des lipides, du cholestérol, de la bilirubine, réaction de Mac Lagan, de Hanger, dosage des protides, étude du rapport A/G, électrophorèse), et par l'examen anatomo-pathologique des ponctions de foie;

D'étudier le retentissement de l'affection sur l'hématopoïèse par l'hémogramme, un médullogramme et la recherche des auto-anticorps anti-érythrocytaires anormaux;

Enfin tous les symptômes particuliers, comme fièvre, ictère, adénopathies, nous ont conduit à des recherches particulières.

Voici quels en sont les résultats:

LA RECHERCHE DES CAUSES ÉVENTUELLES.

L'anamnèse est peu interprétable, vu la difficulté de l'interrogatoire chez des malades qui ne se souviennent pas de leurs malaises et qui ne vivent que dans le moment présent. Il est cependant évident que, dans la grande majorité des cas, ces malades ont présenté dans leur enfance un paludisme, une parasitose intestinale sinon une bilharziose et toutes les agressions communes extérieures que nous pouvons grouper sous le terme le plus général d'agressions dues à l'«environnement».

Si le malade est parvenu à l'âge adulte avec cet héritage morbide certain, il est par contre plus difficile à cet âge de mettre en évidence une ou plusieurs de ces causes alors latentes et apparemment bien supportées par le sujet. C'est ainsi que nos examens systématiques ont pu mettre en évidence les affections suivantes:

1. *Paludisme:*

Recherché sur 3 gouttes épaisses après splénocontraction et à l'occasion de clochers fébriles, nous avons trouvé le *Pl. falciparum* à 24 reprises seulement. Sur ces mêmes gouttes épaisses, nous avons constaté 2 fois la présence de *Filaria perstans;* 1 fois la présence de *Spirocheta duttoni.*

2. *Les tréponématoses:*

34 fois la sérologie des tréponématoses a été trouvée positive, mais il n'existait chez aucun de nos malades des stigmates cliniques authentiques de ces affections et cette réaction sérologique pouvait être assimilée à celles habituellement rencontrées — et dans la même proportion — sous les Tropiques que nous avons coutume d'appeler «cicatrice sérologique».

3. *La bilharziose:*

Elle a été prouvée:

6 fois par l'anamnèse seule qui rapporte la notion d'hématuries dans l'enfance.
5 fois par la cystoscopie seule qui a montré la vessie bilharzienne habituelle.
5 fois par la radiographie de la vessie sans préparation qui a montré le liseré vésical sus-pubien de Pellegrino.

4 fois par l'examen des urines où l'on a décelé la présence d'œufs de *Sch. haematobium.*
3 fois par la ponction du foie qui a ramené un bilharziome intra-hépatique.
1 fois par l'examen de selles qui a montré des œufs de *S. mansoni.*

soit 24 fois la confirmation de l'infection bilharzienne, ce chiffre étant celui assez exact de l'infestation de l'Africain de nos régions.

4. *L'ankylostomiase:*

Elle a été prouvée 18 fois par la découverte des œufs dans les selles.

Toutes ces étiologies purement tropicales ont été mises en évidence, sans que l'on puisse dire qu'elles fournissaient une explication à l'origine de la splénomégalie, car les mêmes pourcentages sont ordinairement trouvés chez les autres malades sans splénomégalie apparente.

5. *Autres causes:*

Nous avons mis en évidence des causes authentiques de splénomégalie. Ce sont:

5 tuberculoses hépatospléniques ou spléniques isolées.
2 typhoïdes.
1 hépatite ictérigène prolongée.
2 trypanosomiases.
14 hémopathies (Hodgkin réticulo-sarcome, leucémie myéloïde, leucémie lymphoïde, Kalher, lymphosarcome).

Ces 24 observations avec étiologies spécifiques et reconnues s'opposent aux 76 autres pour lesquelles nous avons été absolument incapables de porter un diagnostic étiologique précis.

LE BILAN HÉPATIQUE.

Nous avons cherché pour chacun de ces malades à établir un bilan hépatique précis. Voici quels en ont été les résultats:

La lipidémie a été trouvée augmentée 8 fois sur 70.
Le cholestérol a été trouvé élevé 3 fois, abaissé 32 fois, normal 37 fois.
La bilirubine, dosée 92 fois, était présente dans le sang à des taux normaux 59 fois.

10 fois élévation parallèle des 2 bilirubinémies.
9 fois la bilirubinémie directe était seule élevée.
14 fois la bilirubinémie indirecte.

Le Mac Lagan pratiqué 80 fois était normal 16 fois, élevé 64 fois.
Le Hanger floculait 67 fois sur 77 examens, 10 fois s'est relevé normal.
Les protides, dosés 84 fois, étaient abaissés au-dessous de 60 °/₀₀ chez 6 malades, dépassaient le taux de 90 g °/₀₀ 12 fois, étaient à un taux normal 56 fois.
L'électrophorèse, pratiquée 64 fois, donnait un profil électrophorétique à peu près normal chez 4 malades. On notait par ailleurs 60 pics élevés des gamma globulines, 27 pics des béta globulines, 23 pics des alpha globulines.
Le rapport A/G atteignait 0,9 une fois.

16 fois était compris entre 0,5 et 0,9.
26 fois entre 0,3 et 0,5.
10 fois inférieur à 0,3.

La ponction de foie fut pratiquée chez tous nos malades. Elle se révèle indispensable pour juger de l'état anatomique et fonctionnel du foie avant de décider une splénectomie; de plus, elle nous a permis des diagnostics que nous n'aurions pu trouver sans elle, à savoir 4 tuberculoses hépatospléniques et 3 bilharzioses hépatiques.

Voici rapidement les résultats de la lecture de ces biopsies hépatiques que nous devons au Dr. Camain:

Foie de parenchymatose pure:

10 cas, où l'on note seulement quelques modifications discrètes des cellules hépatiques elles-mêmes.

Foie dit «de splénomégalie»:

36 fois, où l'on note un aspect bien particulier de la coupe, qui peut permettre sur simple examen d'affirmer l'existence d'une splénomégalie associée. L'aspect représente constamment une souffrance discrète des hépatocytes, une collagénisation fréquente du précollagène intra-lobulaire, mais surtout un peuplement en cellules rondes des capillaires radiés, qui sont augmentées de volume et hébergent des chapelets caractéristiques de lympho-histio-monocytes.

Foie de fibrose portale ou de fibrose portale et interstitielle associées:

18 fois, où les bandes de sclérose, parfois inflammatoire, coexistent avec une ordonnance normale de l'architecture hépatique, sans réaction adénomateuse du parenchyme.

Foie de cirrhose:

12 fois, où l'on retrouve, associé à la sclérose portale, un bouleversement de l'architecture du foie et des cellules hépatiques en régénérescence active adénomateuse.

Ces deux derniers aspects comportent avec une grande fréquence l'existence de chapelets de cellules rondes peuplant les capillaires radiés et ce phénomène permet là encore de pouvoir porter le diagnostic de splénomégalie. Sur certaine coupe, il est difficile de dire s'il s'agit d'un «foie de splénomégalie» ou d'un foie de fibrose (4 fois), d'un foie de fibrose ou d'un foie de cirrhose (1 fois); ces aspects forment les termes intermédiaires de passage entre ceux des étapes successives d'altération de la glande hépatique.

Nous avons de plus constaté l'existence:

D'un foie infectieux.
D'un foie de réticulo-sarcome.
D'un foie sicklémique.
De 3 foies bilharziens.
De 2 foies palustres.
De 4 foies tuberculeux.
De 7 foies cardiaques.

Parmi ces foies cardiaques, un seul s'observait avec un gros cœur de myocardie, les 6 autres ne s'accompagnaient d'aucun signe clinique, électrique ou radiologique de souffrance cardiaque, et étaient le témoin de foie anoxique, vasculaire sans cardiopathie sur lesquels nous reviendrons dans le détail au chapitre de la discussion.

ÉTUDE DE LA CIRCULATION PORTALE.

La *pression portale* a été recherchée par la méthode de la ponction splénique transpariétale dans 34 observations. Elle a été trouvée normale chez 15 malades; elle dépassait le chiffre de 15 cm d'eau pour atteindre des chiffres très élevés comme 45 cm d'eau chez 19 malades.

La *splénoportographie* a été pratiquée 31 fois: 23 fois, elle s'est révélée normale sinon un aspect de dilatation de la veine porte et une flexuosité de la veine splénique; 8 fois anormale et l'on notait une fois un blocage de la veine porte; 7 fois un reflux porto-cave important avec apparition de collatérales.

ÉTUDE DU RETENTISSEMENT SANGUIN DE LA SPLÉNOMÉGALIE.

L'étude hémato-immunologique que nous devons au D^r^ Baylet nous a montré:

Dans le sang périphérique:

12 % de sangs normaux.
4 % de sangs anémiques purs.
23 % de pancytopénies.

23 % de syndromes sélectifs anémico-neutropéniques.
18 % de syndromes sélectifs anémico-lymphopéniques.
11 % d'anémies avec lymphocytose.
5 % de neutropénie pure.
4 % de neutropénie et lymphopénie associées sans anémie.
4 % de sang leucocytosique.
L'anémie était dans 55 % des cas isochrome, 26 % des cas hypochrome, 18 % des cas hyperchrome.

Parmi les anémies hypochromes, on constate une microcytose dans 14 % des cas, une normocytose dans 11 % des cas.

Parmi les anémies hyperchromes on note 7 % de microcytose, 11 % de macrocytose.

Dans la moelle osseuse:

41 % de moelles érythroblastiques.
34 % de moelles granulopéniques.
13 % de moelles lymphocytiques.
12 % de moelles normales.

Les examens immunologiques n'ont pas décelé la présence d'auto-anticorps anti-érythrocytaires complets ou incomplets sur 60 examens.

En résumé, le retentissement sanguin de la splénomégalie est donc marqué avant tout par un aspect dit d'«hypersplénisme» fréquent, avec sang périphérique pauvre (anémie et neutropénie), la pénurie étant soit globale, soit dissocié, mais avec moelle riche, régénérative pour les globules rouges, peu régénérative pour les globules blancs.

*

Voilà les faits constatés. Il nous faut à présent discuter le problème sous quatre aspects:

1° Tout d'abord sous l'*angle pathogénique,* analyser les symptômes un à un et essayer de les expliquer pour chaque malade en particulier;

2° Analyser ensuite sous l'*angle nosologique* chaque observation une à une et les grouper dans l'ensemble des cas;

3° Envisager ensuite la maladie sou l'*angle évolutif et thérapeutique;*

4° Dans un dernier chapitre enfin, dégager les quelques idées générales qui nous paraissent devoir être retenues.

LES SIGNES GÉNÉRAUX.

Nos splénomégaliques présentent ordinairement un état général satisfaisant: 17 d'entre eux présentaient un état général médiocre et l'analyse de ces cas nous a montré qu'il s'agissait:

De 2 typhiques.
De 5 tuberculoses spléniques.
De 3 cirrhotiques.
De 7 hémopathiques:

1 Hodgkin.
1 leucémie myéloïde.
1 sicklanémie.
2 réticuloses malignes.
1 lymphosarcome splénique.
1 leucémie lymphoïde à forme splénique pure.

L'existence d'un mauvais état général chez un sujet atteint d'hépatosplénomégalie paraît donc devoir localiser le problème du diagnostic étiologique à des affections bien précises.

LES ŒDÈMES.

Nous les avons constatés chez 31 malades; leur importance était variable:

Soit œdème des membres inférieurs isolés (10 fois).
Soit ascite isolée (16 fois).
Soit anasarque (5 fois).

Nous avons retrouvé 16 fois une cause à la présence d'œdème:

2 fois il s'agissait d'œdèmes cardiaques.
14 fois, l'altération hépatique les expliquait et parmi les malades de ce groupe, 5 présentaient une pression portale élevée, 3 présentaient une hypo-protidémie, aucun ne présentait d'altération rénale (urée 99 fois normale, 1 seul malade présentait une azotémie à 0,60 g °/₀₀). Quant aux 15 autres malades, il ne nous a pas été possible de déterminer la cause de leurs œdèmes.

LES ICTÈRES.

Retrouvés chez 10 malades, ils se sont toujours accompagnés d'une bilirubinémie indirecte élevée, 8 fois d'une hyper-bilirubinémie directe. Une seule fois cet ictère paraissait être celui de l'hépatite ictérigène. Les 9 autres malades qui présentaient plus un subictère qu'un ictère, étaient soit des cirrhotiques, soit des hémopathiques, et deux présentaient un paludisme en activité.

LES ADÉNOPATHIES.

Retrouvées chez 16 malades. Importantes chez 3 hémopathiques, elles étaient discrètes chez les autres malades, parmi lesquels on note la présence de 1 trypanosomiase, 1 sicklanémie, 2 tuberculoses spléniques. Les 9 autres malades étaient porteurs de ganglions anormaux banalement inflammatoires.

LES GROS CŒURS.

Nous avons vu que 15 de nos malades présentaient un gros cœur de myocardie: il s'agissait de 7 cœurs anémiques irréversibles, de 2 gros cœurs de trypanosomiases réversibles, de 6 hypertrophies cardiaques idiopathiques fréquemment rencontrées sous les Tropiques.

LES CARACTÈRES DE LA RATE (taille, contractilité, sensibilité).

Ils ne fournissent aucun élément intéressant et ne permettent absolument pas de préjuger de l'importance de l'atteinte hépatosplénique. Une grosse rate volumineuse non contractile a peu de chance toutefois de se modifier et constitue à nos yeux une indication de splénectomie, car elle est gênante pour le malade.

LES CHIFFRES DE PRESSION PORTALE.

Ils ne donnent eux aussi aucune indication précise sur l'état hépatosplénique. Cependant une pression portale élevée est volontiers retrouvée en cas d'hématémèse, d'ascite isolée, de fibrose ou cirrhose hépatique, de rate non contractile; mais outre qu'une pression portale normale peut être constatée chez un malade qui présente ces signes, on peut constater de plus une pression portale élevée chez un sujet qui ne présente ni hématémèse, ni fibrose hépatique, ni ascite et dont la rate demeure contractile. Nous avons pu constater également sur les clichés après splénoportographie que la pression portale est basse lorsqu'une circulation collatérale s'est développée. En définitive, connaître le chiffre de la pression portale nous paraît d'une utilité discutable.

LES ASPECTS ANATOMO-PATHOLOGIQUES DU FOIE.

Ils sont évidemment utiles à connaître pour juger de l'état de la glande hépatique, mais fournissent également des indications précieuses sur l'état de la rate, de la circulation portale, et sur une étiologie éventuelle.

Les *foies de parenchymatose pure* se voient volontiers lorsque le malade présente une sérologie syphilitique positive, est sicklémique, et qu'il existe une hépatomégalie ou une volumineuse rate.

Le *foie dit «de splénomégalie»* est souvent un gros foie et se voit volontiers dans l'ankylostomiase. La rate est alors modérément hypertrophiée.

Le *foie de fibrose* est ordinairement un foie de taille subnormale, accompagne une splénomégalie modérée, mais coexiste volontiers avec une pression portale élevée, une ascite isolée ou l'apparition d'œdème et d'un subictère.

Le *foie de cirrhose* est encore un gros foie qui se rencontre dans les splénomégalies très développées, et qui coexiste volontiers avec une ascite isolée et une sérologie syphilitique positive.

Le *foie cardiaque* est un gros foie, avec splénomégalie modérée (n° 3), ordinairement sans ascite, ni ictère, qui se rencontre volontiers avec des chiffres très élevés de pression portale et lorsque le sujet est paludéen et anémique, parfois bilharzien ou tuberculeux. Il est à noter à ce sujet que le foie de nos malades porteurs d'une cardiomégalie étaient selon un ordre de fréquence décroissant, des foies de parenchymatose, puis de fibrose et enfin bien loin après des foies cardiaques. La notion de foie cardiaque chez un porteur de splénomégalie ne doit donc pas aiguiller uniquement les recherches étiologiques vers une défaillance cardiaque, mais aussi vers une tuberculose splénique, un paludisme, une anémie, voire une bilharziose au début. Nous pensons qu'il s'agit alors de foie anoxique et nous ne gardons le terme de foie cardiaque qu'à cause de son aspect identique sur coupe.

Nous faisons remarquer enfin qu'il ne s'agissait pas de syndrome de Budd Chiari, et que cet aspect congestif centro-lobulaire bien particulier a été retrouvé à deux ponctions successives afin d'éviter tout artéfact dû au traumatisme de l'aiguille.

CARACTÈRES ÉVOLUTIFS DES HÉPATOSPLÉNOMÉGALIES

Tous nos malades ont été traités et suivis dans le service durant une période de un mois environ. Nous avons évidemment traité les facteurs étiologiques éventuels (syphilis, tuberculose, hémopathies, paludisme, parasitose, etc.), et nous avons pratiqué pour tous des perfusions veineuses lentes d'un «cocktail» en usage dans le service contenant de l'extrait de foie, de la cortisone, des vitamines, des protecteurs hépatiques. Lorsque la rate était gênante par son volume et sa sensibilité, lorsqu'il existait un syndrome d'hypertension portale, nous avons proposé une splénectomie au malade.

A la sortie du service, tous nos malades ont reçu un traitement anti-palustre à suivre à la maison et ont été convoqués à notre consultation externe. De nombreux malades ont pu être ainsi suivis durant une période plus ou moins longue au cours de ces deux années. Voici nos constatations:

10 malades sont décédés.

65 malades ont été améliorés et ont quitté l'hôpital en excellent état général, sans que leur splénomégalie ait regressé pour autant.
18 malades ont dû être réhospitalisés, et parmi eux 3 sont dans un état stationnaire.
9 malades ont vu rétrocéder leur hépatosplénomégalie dans les mois qui ont suivi l'hospitalisation.
17 malades enfin ont été splénectomisés, dont 12 au cours de leur deuxième hospitalisation.

Étudions en particulier chacune de ces constatations:

LES DÉCÈS.

Parmi les 10 malades décédés, on distingue:

1 malade atteint de réticulo-sarcome splénique.
1 malade atteint de leucémie lymphoïde, porteur d'une cirrhose hépatique avec ébauche de cancérisation.
1 malade atteint de maladie de Hodgkin avec aspect leucémique du foie.
1 malade atteint de leucémie lymphoïde à forme splénique pure, décédé 3 jours après splénectomie.
1 malade décédé d'une hématémèse foudroyante.
1 malade anémique grave.
1 malade typhique.
1 malade atteint de tuberculose splénique ulcéro-caséeuse, décédé après splénectomie.
1 malade décédé après un coma de courte durée et représentant les stigmates biologiques de la trypanosomiase.

Tous ces décès étaient prévisibles et ne méritent aucun commentaire.

AMÉLIORATION.

63 malades, après leur séjour dans le service, ont repris une activité normale et un bon aspect général. Nous n'avons pas de recul

suffisant pour préjuger de l'évolution réelle de leur affection, du devenir de leur splénomégalie, de l'évolution ultérieure vers des complications relevant de l'hypertension portale, de l'installation d'une cirrhose hépatique par exemple, de la gravité chez ces malades d'une rencontre avec le virus de l'hépatite ictérigène. Quoi qu'il en soit, ces malades se conduisent dans la vie comme les individus apparemment normaux qui les entourent et leur grosse rate demeure le seul symptôme pathologique.

18 malades ont dû être réhospitalisés *pour rechute* des symptômes qui avaient motivé leur première hospitalisation. Parmi ceux-ci, 3 ont acquis un état de guérison semblable à celui des malades du groupe précédent; 3 malades demeurent dans un état stationnaire sans que l'on puisse noter une amélioration, à savoir une tuberculose splénique, 2 malades de Kalher; 11 de ces malades enfin ont été splénectomisés.

9 malades ont présenté une *guérison clinique apparemment complète* de leur affection, à savoir:

1 malade typhique.
3 malades atteints de tuberculose splénique.
1 malade atteint de trypanosomiase.
4 malades porteurs d'hépatosplénomégalies spontanément régressives en 3 à 6 mois.

Ces derniers malades porteurs de grosse rate contractile et de foie «de splénomégalies» avaient reçu en vain le traitement habituel, en particulier un traitement antipalustre sans que leur hépatosplénomégalie ait regressé. Revus en consultation 3 à 6 mois plus tard, la taille du foie et des rates était sub-normale. Nous n'avons aucune étiologie à proposer pour ces malades particuliers, et nous avons été surpris de l'évolution. Un bilan pratiqué alors s'est révélé en tous points semblable au premier bilan pratiqué à l'entrée dans le service. Il semble donc bien que la taille de la rate soit un épiphénomène, et la maladie indépendante de la taille de l'organe.

LES SPLÉNECTOMIES.

17 malades ont été splénectomisés: il s'agissait de rates pesant de 1 kg à 2,800 kg. Les indications ont été les suivantes:

Tuberculose splénique, 2 fois.
Hématémèse, 1 fois.
Lymphosarcome de la rate, 1 fois.
Splénomégalie bilharzienne, 1 fois.
Leucose lymphoïde, 1 fois.
Énorme rate gênante, 11 fois.

Pour ces derniers cas, nous n'avons pratiquement pas tenu compte du chiffre de pression portale, de l'état du foie, des stigmates d'hypersplénisme. Certes, une pression portale élevée, un foie en voie d'altération, des signes évidents d'hypersplénisme invitaient à l'intervention, mais la gêne de l'hypocondre gauche que ressentait de malade a constitué pour nous l'indication majeure. L'examen anatomopathologique de ces rates nous a montré qu'il s'agissait de:

Rate congestive, 1 fois.
Rate congestive avec réaction monocyto-réticulaire, 1 fois.
Rate congestive avec réaction macrophagique et discrète fibroadénie, 1 fois.
Rate de réticulose avec hyperactivité des formations lymphoïdes et artérite, 1 fois.
Rate fibro-congestive, 2 fois.
Rate fibro-congestive avec réaction H.L.M.[1], 2 fois.
Rate fibreuse, 2 fois.
Rate fibreuse avec nodules de Gandy Gamna et réaction préleucosique lymphoïde, 1 fois.

Ces aspects ne sont évocateurs d'aucune étiologie particulière. Tout au plus a-t-on pu établir une relation entre la présence d'une hypertension portale élevée et la présence d'artérite splénique, celle d'une maladie de Hansen et d'une réaction macrophagique splénique. Enfin, fait à signaler, le réticulum splénique est toujours très fortement majoré.

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[1] Histio-lymphomonocytaire.

Nous avons pu suivre pour ces malades les effets de l'intervention sur l'évolution de leur maladie et pour 5 d'entre eux, étudier l'aspect anatomique du foie par ponction de foie et l'aspect hématologique:

Les 3 décès sont dûs à 2 thromboses portes post-opératoires et à un coma hépatique.

2 malades n'ont tiré aucun bénéfice apparent de la splénectomie; il s'agit d'un lympho-sarcome de la rate et d'une tuberculose splénique.

Les 12 autres malades ont été améliorés par l'intervention, ils ont repris du poids, les œdèmes et l'ascite ont disparu et les résultats ont été d'autant plus spectaculaires que l'état antérieur du foie était moins grave.

Les bilans post-splénectomies plusieurs mois après l'intervention nous ont fourni les constatations suivantes:

L'étude des biopsies hépatiques pratiquées par le D^r^ Camain plusieurs mois après la splénectomie ont montré, à trois reprises, une amélioration des signes de souffrance des hépatocytes, et une accentuation des signes de mésenchymatose; à 2 reprises (1 cirrhose et 1 tuberculose splénique) un état identique à celui des biopsies hépatiques pratiquées avant la splénectomie. Chez 3 malades sur 5, tout se passe donc comme si la splénectomie améliorait les conditions de vie de la cellule hépatique, et entraînait une hypertrophie du S.R.E. hépatique.

L'étude hématologique que nous devons au D^r^ Baylet a montré les faits suivants:

1. *Action de la splénectomie sur l'érythropoïèse*: après le bouleversement qui suit l'intervention, le chiffre des globules rouges s'est rétabli à un taux ancien. Dans la moelle, on constate une normalisation mais sans modifications importantes. L'activité fonctionnelle médullaire mesurée par le taux des réticulocytes revient identique à ce qu'elle était durant la période précédant l'intervention. Il apparaît par contre des formes à vestiges nucléaires traduisant le passage de

globules rouges inachevés dans le sang par trouble de la maturation nucléaire (corps de Jolly), par trouble de la synthèse de l'hémoglobine. Il existe une augmentation de la résistance globulaire osmotique maxima.

2. *Action sur les plaquettes:* dans le sang il existe toujours une très nette augmentation des thrombocytes, mais cette thrombocytose diminue progressivement pour atteindre un chiffre sensiblement normal. Dans la moelle le taux des mégacaryocytes est très augmenté; on compte de 9 à 30 mégacaryoblastes ou mégacaryocytes pour 10 000 cellules nucléées (chiffre normal: 5).

3. *Action sur la leucopénie:* dans le sang on constate une leucocytose importante qui également diminue avec le temps. Il s'agit soit d'une neutrophilie, soit d'une lymphocytose et souvent les deux associées. Le taux des éosinophiles est très variable. La formule leucocytaire est normale. Dans la moelle, le phénomène le plus important et le plus constant est l'augmentation du nombre des métamyélocytes et des polynucléaires, par rapport aux examens pratiqués avant la splénectomie. Par contre, le taux des lymphocytes est très variable. Dans 2 observations, le taux des plasmocytes a augmenté (9 à 10 %) 3 et 5 mois après la splénectomie. Il n'y a pas d'augmentation du nombre des cellules réticulaires.

En résumé, il semble que la splénectomie, après d'importantes variations, permet une distribution plus normale des leucocytes, en particulier par l'augmentation du nombre des polynucléaires circulants, par maturation ou libération normales des granulocytes médullaires. Elle entraîne essentiellement d'une manière permanente un trouble de la maturation nucléaire des normoblastes et de la synthèse de l'hémoglobine.

CONCLUSIONS

Ces faits d'observations nous permettent de tirer des conclusions justes sinon précises ou exactes:

1° Il existe chez l'adulte en Afrique des hépatosplénomégalies d'étiologie pure, que l'on peut voir regresser sous l'action du traitement spécifique. C'est le cas des splénomégalies tuberculeuses, typhiques, cardiaques, de certaines hémopathies.

2° Il existe également des hépatosplénomégalies secondaires à des causes multiples, mais où une étiologie prédominante palustre, bilharzienne ou syphilitique par exemple est décelée par le succès du traitement spécifique. Il s'agit là des étiologies classiques.

3° Il existe enfin des hépatosplénomégalies de cause inconnue, les plus nombreuses. On a invoqué à leur sujet le rôle déterminant d'un facteur nutritionnel ou d'un facteur infectieux, difficiles à préciser. Le facteur parasitaire existe sans nul doute, mais le malade ne garde plus alors que le souvenir lointain d'une infection palustre, d'une hématurie bilharzienne. A l'âge adulte ces infections sont éteintes; le sujet est prémuni à l'endroit du paludisme, les lésions bilharziennes se sont calcifiées. Enfin l'incidence de certaines affections, comme la trypanosomiase par exemple, n'est pas négligeable, mais encore obscure. En fait, il est remarquable qu'il s'agisse toujours d'affections à détermination réticulo-endothéliale prédominante. La preuve en est dans les résultats de l'exploration fonctionnelle spléno-hépatique et dans l'altération constante du profil électrophorétique.

Les expériences, sur l'animal, de blocage du S.R.E. par des molécules expérimentales ont montré que les infiltrations de cellules rondes vont de pair avec une altération du profil électrophorétique, et ces altérations sont d'une manière certaine le témoin de la présence dans le sang de macromolécules d'origine endogène ou exogène. Ces phénomènes sont à eux seuls suffisants pour produire une hépatosplénomégalie et pour l'entretenir. Or tout peut se trouver à l'origine de ces altérations particulières du foie et de la rate de l'Africain; tout ce qui lèse le système porte, tout ce qui lèse la rate, tout ce qui représente la pathologie courante de l'Africain peut conduire à ces réactions qui sont des réactions spécifiques du S.R.E. L'importance du foie a été exagérée par rapport au système réticulo-endothélial; on a créé une pathologie hépatique africaine en attribuant au foie une foule de choses qui ne lui revenaient pas. Le point important de l'étude de ces hépatosplénomégalies — si on les ramène à leur plus simple expression — est que l'on peut en distinguer deux types:

> Celles qui sont réversibles, soit sous l'action d'une thérapeutique, soit spontanément pour des raisons qui nous échappent complètement;

> Celles qui sont irréversibles et qui posent le problème de la splénectomie, car à ce stade les choses se passent déjà pratiquement comme si la rate était exclue, la mobilisation des hémo-histioblastes de tout l'organisme, notamment au niveau du foie, étant le témoin de la réaction du mésenchyme actif en présence d'un blocage plus ou moins complet du S.R.E. splénique.

Nos conclusions sont basées sur nos observations personnelles. Elles ne sont pas cependant isolées dans le monde. Des constatations identiques ont été en effet rapportées ces temps derniers par Lanzo, en Erythrée; Kirk, en Égypte; Chaudhuri, dans les Indes. Nous retrouvons chez ces auteurs, sous les termes de «splénomégalie tropicale essentielle», «splénomégalie chronique», la description exacte de nos hépatosplénomégalies de cause inconnue: les lésions hépatiques, les altérations sanguines, le même défaut d'étiologie sûre, se retrouvant fidèlement. La convergence de tels résultats simultanément retrouvés par les méthodes modernes d'investigation, dans des régions aussi éloignées que les nôtres, est pour notre conception d'un intérêt majeur. Il ne peut plus s'agir en effet de splénomégalie palustre, de splénomégalie bilharzienne, mais d'une entité clinique réelle, d'une réticulo-endothéliopathie hépatosplénique.

RÉSUMÉ

La rate des Africains est rarement normale. Sur 100 malades africains adultes observés, porteurs de splénomégalie manifeste, 61 avaient un gros foie, 31 des œdèmes, 16 des adénopathies, 15 un gros cœur, 10 un ictère.

La cause de ces splénomégalies a été isolée 24 fois (5 tuberculoses, 2 typhoïdes, 1 hépatite ictérigène prolongée, 2 trypanosomiases, 14 hémopathies). 76 fois aucun diagnostic étiologique précis n'a pu être posé. Parmi ces malades certains présentaient un paludisme, une tréponématose, une bilharziose, une ankylostomiase. Mais ces affections sont retrouvées avec une fréquence identique chez les sujets sans splénomégalie apparente. Le bilan hépatique montre des signes constants d'hyper-réticulisme. La ponction biopsie hépatique montre soit une atteinte parenchymateuse discrète du foie, soit une atteinte mésenchymateuse signée fréquemment par la présence de cellules rondes dans les capillaires radiés, moins souvent par des travées de fibrose. L'étude des pressions portales et des spléno-

portographies a été pratiquée, mais ne présente aucune utilité. Dans le sang il existe les signes d'hypersplénisme classique. 17 splénectomies ont été faites chez des malades qui étaient gênés par la taille de leur rate.

Il semble donc exister chez l'Africain adulte d'A.O.F.: 1° des hépatosplénomégalies d'étiologie pure; 2° des hépatosplénomégalies secondaires au paludisme, à la bilharziose; ce sont les causes classiques; 3° des hépatosplénomégalies de cause inconnue, les plus nombreuses réversibles ou irréversibles, qui réalisent une entité clinique réelle, une réticulo-endothéliopathie hépatosplénique.

NUTRITIONAL STUDIES ON BREAST MILK OF NIGERIAN WOMEN

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The main problem facing paediatricians in Africa is that of ensuring that babies are adequately fed in the first two years of their lives. In most cases, breast milk is the diet of choice because (*a*) it is easy to administer and (*b*) it obviates the difficulty of rendering the feeding conditions relatively sterile.

Although lactation and breast feeding usually follow an uncomplicated course in most Nigerian mothers, the actual output of milk is not copious, especially after the first month or two (Bassir, 1956).

Most attempts to improve the nutrition of African and Indian babies have taken the form of supplementation of the infant's diet with milk products (Autret and van Deen, 1955) or with leguminous powder (Dean, 1953; Subrahmanyan *et al.*, 1954, 1957). However, the former is expensive and easily infected, and the latter may upset the digestive system of the babies and cause diarrhoea (Ademola, 1958).

It was, therefore, considered worthwhile to find out whether the output of milk of Nigerian women could be increased by supplementing their staple diets with a protein-rich leguminous flour.

EXPERIMENTAL SUBJECTS

A group of lactating mothers drawn at random from the attendants at the Infant Welfare Clinic in Lagos were paired according to setage of lactation and social status. One lot served as control, while the other received a daily supplement of 30 g soya bean flour per person. The legume was added to the customary diet of the women, and consumed in any manner the subjects chose.

SOYA FLOUR

Dried beans grown on the Middle Belt of the Nigerian plateau were autoclaved for two hours at 120°C and then put through an expelling machine. The product obtained was a whitish powder with a slightly sweet taste. This was distributed to the experimental subjects in 2 lb. air-tight tins. Refills were given every four weeks. The results of analysis of the soya flour are shown in Table 1.

TABLE 1 — *Nutrients in the soya flour*

Nutrient.	Amount (g/100 g)
Protein	47.5
Fat	18.4
Carbohydrate (as glucose)	24.6
Water	9.11
Calcium	0.210
Ash	—

DIETS

The mothers taking part in this experiment were on diets composed of locally-produced foodstuffs. The diets were usually adequate calorifically. Their protein and calcium contents were, however, below the recommended daily dietary allowances of the Food and Nutrition Board of the U.S. National Research Council for lactating mothers. The daily intake of nutrients of the experimental subjects are shown in three typical diets, and compared with the recommended allowances (Table 2).

TABLE 2 — *Three typical diets of the experimental subjects*

(1) Fresh red pepper 85 g; meat 225 g; bread 114 g; rice 85 g; gari (cassava farina) 171 g; yam 450 g; palmoil 86 g; crayfish 10 g	
Total calories	3,042
Total proteins	81 g
Animal protein	49.1 g
Calcium	332 mg
(2) Fresh red pepper 86 g; fish 57 g; meat 57 g; rice 121 g; beans 57 g; gari 228 g; yam 114 g; palmoil 70 g	
Total calories	2,620
Total proteins	63 g
Animal protein	19.1 g
Calcium	353.4 mg
(3) Fresh pepper 114 g; meat 225 g; gari 342 g; yam flour 342 g; palmoil 70 g; crayfish 8 g	
Total calories	2,703
Total proteins	64.4 g
Animal protein	41.5 g
Calcium	416.8 mg
Recommendations of the U.S. National Research Council: Food and Nutrition Board (1948)	
Calories	3,000
Protein (g)	100
Calcium (g)	2.0

CLINICAL PROCEDURE

Previous experiments had shown that the secretion of milk by Nigerian mothers suffered a diurnal variation with maximal output in the middle of the morning (Bassir, 1957). The mothers were made to breast feed their babies for ten minutes, commencing at 8.50 a.m. At 9 a.m. the breasts were drained by manual expression.

The babies were then separated from their mothers. At 12.50 p.m. they were weighed and put to the breast. Some drops of milk were expressed into a container for later analysis and suckling from both continued till 1.10 p.m. The babies were then test-weighed again.

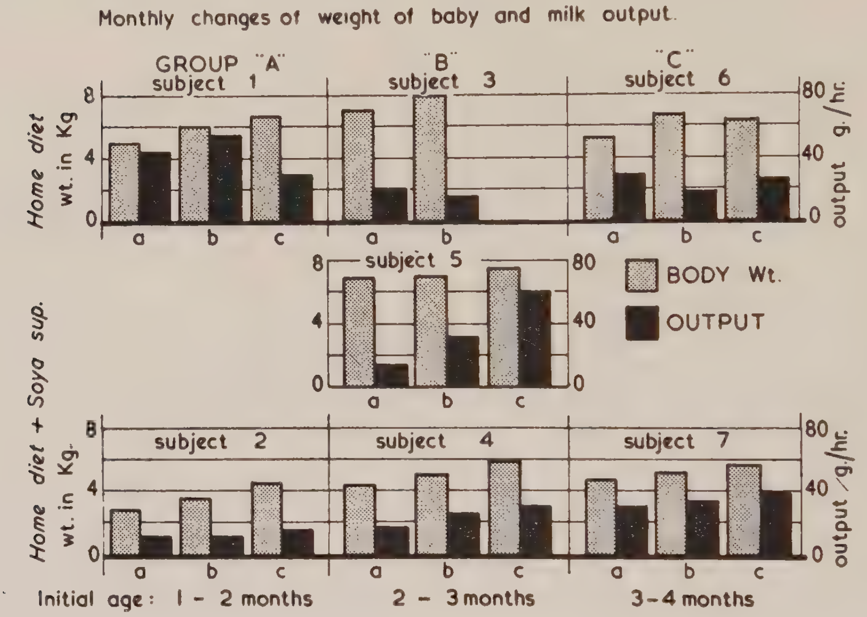


Fig. 1

The output of milk for the four-hour period was thus obtained. The whole procedure was repeated every four weeks. The monthly changes of the weights of some of the babies and the milk outputs of their mothers are illustrated in Fig. 1.

ANALYTICAL METHODS

The milk samples obtained from the subjects were analysed for total nitrogen, non-protein nitrogen, «cream», calcium and phosphorus by methods which have been described in a previous paper (Bassir, 1956*a*). The procedure followed in the determination of phosphatase was that described by King (1951).

Data for Fig. 1

Monthly changes of weight of baby and milk output

	Weight of baby (kg)			*Hourly output of milk (g)*		
	Group A					
			(1)			
On home diet	(a) 4.74	(b) 5.74	(c) 6.36	(a) 34	(b) 45	(c) 28
			(2)			
On home diet plus soya	(a) 2.94	(b) 3.53	(c) 4.18	(a) 13	(b) 13	(c) 16
	Group B					
			(3)			
On home diet	(a) 6.72	(b) 7.95		(a) 21	(b) 17	
			(4)			
On home diet plus soya	(a) 4.42	(b) 4 99	(c) 5.72	(a) 18	(b) 25	(c) 30
			(5)			
On home diet plus soya	(a) 6.72	(b) 6.73	(c) 7.35	(a) 15	(b) 29	(c) 61
	Group C					
			(6)			
On home diet	(a) 5.67	(b) 6.61	(c) 6.19	(a) 28	(b) 18	(c) 25
			(7)			
On home diet plus soya	(a) 4.71	(b) 5.25	(c) 5.34	(a) 15	(b) 35	(c) 38

RESULTS

Table 3 shows that the constituents determined do not indicate a statistical change in the composition of the milk secreted after supplementing the mothers' diets with soya flour. However, the mean

TABLE 3 — *Variations in the milk constituents during the period of supplementary feeding*

Identification number	Name	Cream (%)			Total nitrogen (%)			Non-protein nitrogen (mg/100 ml)			Calcium (mg/100 ml)			Phosphorus (mg/100 ml)			Phosphatase (K-A units/100 ml)		
		1st month	2nd month	3rd month	1st month	2nd month	3rd month	1st month	2nd month	3rd month	1st month	2nd month	3rd month	1st month	2nd month	3rd month	1st month	2nd month	3rd month
1	Fatimotu . .	1.3	4.5	6.5	0.33	0.17	0.16	61	45	42.5	17.2	19.1	22.5	8.5	17.0	15	5.7	5.9	18.3
2	Sariyu . . .	3.5	4.5	2.2	0.25	0.15	0.19	55	65	40	13	11.2	13.5	12	25.1	12.5	10.5	12.5	11.2
3	Raheem Ban	1.1	6	2.2	0.21	0.22	0.22	44.5	45	50.5	18.4	44	22.8	8.6	20.5	10.5	7.3	11.7	9.6
4	Musendiku. .	1.5	2.2	2	0.19	0.21	0.13	73	50	45.5	20.6	16	22.3	23.2	26.5	20.5	12.9	13	14.5
5	Olatunji . . .	8.5	9.5	17	0.28	0.24	0.13	45	44.5	50	22.2	16	11.3	13.8	10.8	23.1	11.8	11.8	13.3
6	Sunday . . .	2.5	3	3.5	0.30	0.13	0.27	47	50	55	16.8	13.5	15	13.7	13.2	26	15.0	13.5	14
7	Afusatu . . .	1.5	2.0	2.1	0.19	0.21	0.25	55	47	45	22.5	17.5	15.5	12.5	24	11.5	8.3	21	7.1
	Mean. . . .	2.9	4.7	5.1	0.25	0.19	0.19	54.5	49.5	47	18.9	19.5	17.6	13.1	19.5	17	10.1	12.8	12.6

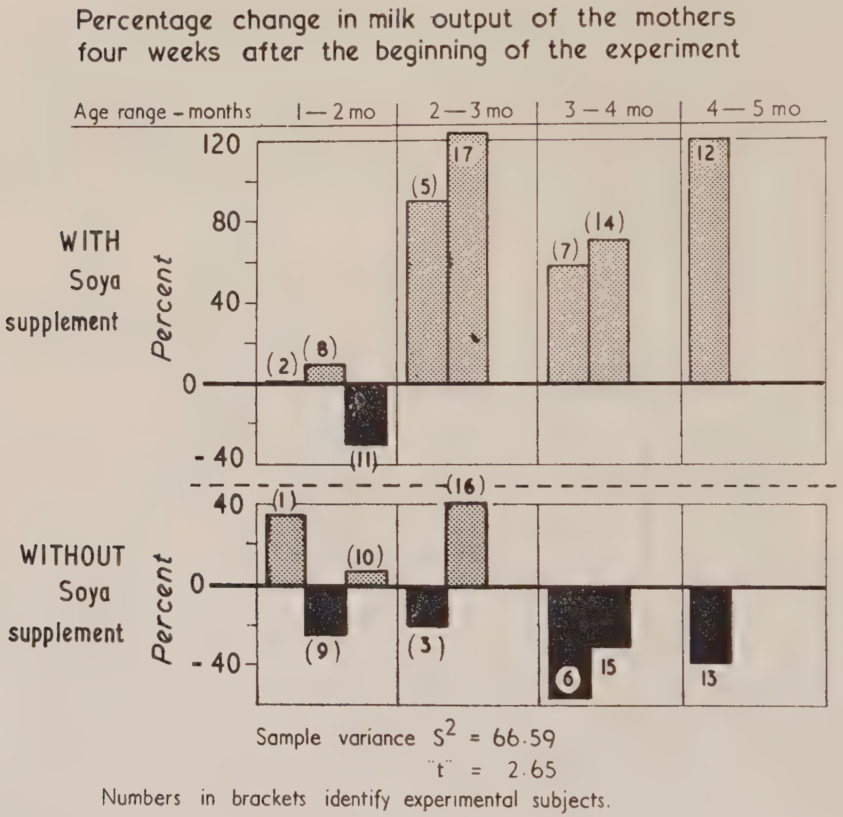
«cream» values are probably significantly different. Table 4 and Fig. 2 indicate the variation in milk output of the mothers after receiving a daily supplement of soya flour for four weeks. When compared according to the stage of lactation, the milk output of those women receiving the soya supplement can be seen to be statistically higher (at the 5 % level) than that of the subjects who took only their customary diets.

TABLE 4 — *Effect of daily supplementation on milk output after one month*

(The numbers in brackets indicate the experimental subjects)

Diet	Variation in output (% of initial weight)			
	Initial age ranges (months)			
	1–2	2–3	3–4	4–5
With soya supplement	0 (2) + 9.5 (8) — 29.4 (11)	+ 93.3 (5) + 125 (17)	+ 57.1 (7) + 72.2 (14)	+ 120 (12)
Without soya supplement	+ 33 (1) — 24.2 (9) + 5.7 (10)	— 19.1 (3) + 43 (16)	— 55.5 (6) — 32 (15)	— 37.5 (13)

Sample variance (S^2) = 66.59.
Students «t» = 2.65.
Difference between the 2 dietary groups probably statistically significant.



Percentage change in milk output of the mothers four weeks after the beginning of the experiment

Sample variance $S^2 = 66.59$

"t" = 2.65

Numbers in brackets identify experimental subjects.

Fig. 2

DISCUSSION

The results presented in this paper suggest that the use of locally-produced leguminous foodstuff as a major additional item of diet of the poorer classes of Nigerian nursing mothers could result in an increased output of breast milk to provide adequately for a growing baby in the first year of life. Since the fall-off of milk secretion of Nigerian women towards the end of the first year and in the second year follows a smooth curve (Bassir, 1956), it may be that these results, which have been obtained with mothers in the first few months of lactation, are applicable to the later stages. Further investigation with women in their second year of lactation are planned.

Gopalan (1957) obtained increased output of milk by supplementing the diet of poor Indian women with skim milk, and the total nitrogen content of the milk did not seem to have been affected. When the dietary supplement provided 30 g of protein a day, the growth performance of the infants was better than that of the group whose mothers fed entirely on their ordinary diets.

Bartchenko (1957) supplemented the diets of Russian mothers of modest socio-economic circumstance with a mixture of yeast, liver, etc., and obtained significant increase both in the amount of milk produced and in some of the mineral elements determined.

The available evidence on the influence of diet on the composition of milk is inconclusive. Escudero and Pierangeli (1943) fed two groups of lactating mothers on diets of differing quality and obtained a higher output of milk with the better diet. Gunther and Stania (1951) noted a lower nitrogenous content of milk from their poorly nourished subjects as compared with that of English mothers. However, both Antonov (1947) and Smith (1947) did not observe significant modifications in the nitrogenous contents of breast milk during severe food shortage in Leningrad and Holland, respectively. Antonov is of the opinion that only the quantity of milk and the duration of lactation are influenced by dietary restriction.

This view is in agreement with the experimental findings described in this paper.

SUMMARY

1) Eighteen Nigerian mothers in the first six months of lactation were selected at random and used in a feeding experiment lasting three months, with a view to assessing the value of supplementing their home diets with 30 g of soya flour daily.

2) The women were paired according to stage of lactation and socio-economic class. One group received the soya in addition to their ordinary food.

3) A probably significant increase in the output of breast milk was obtained after four weeks in the women feeding on the soya flour. This increase was more marked after eight weeks.

4) The composition of the milk was not significantly altered during the course of the experiment. The constituents estimated were total nitrogen, non-protein nitrogen, «cream», calcium, phosphorus, and phosphatase.

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My thanks are due to the Head of the Federal Institute of Applied Technical Research, Lagos, for generously providing the soya bean meal.

I am also indebted to Dr. Tunji Adeniyi-Jones, Miss M. A. Clarke, and other members of the Lagos Infant Welfare Clinic for invaluable assistance in the clinical aspect of this work.

GENERAL DISCUSSION

Dr. J. Sénécal (Dakar — A.O.F.) — Je voudrais commenter rapidement la communication présentée par le D^r Bassir, qui a eu le mérite de s'attaquer au difficile problème des variations de la sécrétion lactée en fonction de l'alimentation. Il semble admis maintenant que le lait se modifie peu dans sa composition et que les variations interviennent principalement dans la quantité sécrété chaque jour.

Mais comment apprécier cette quantité? Il faut hospitaliser la femme, supprimer l'alimentation à la demande pour la remplacer par des tétées régulièrement espacées; il faut encore séparer la mère de son enfant et, pour faire des analyses, effectuer un prélèvement manuel. Tout ceci constitue un certain nombre de chocs psychologiques qui dans l'ensemble diminuent la sécrétion.

Nous avons mesuré l'action de ces différentes perturbations et nos résultats ont, depuis, été confirmés par De Maeyer: les résultats peuvent aisément varier du simple au double. C'est dire la difficulté de mettre au point une bonne technique pour mesurer le volume de la sécrétion lactée.

Mais je voudrais surtout discuter maintenant le problème de santé publique que pose cette communication. L'accord est unanime sur la valeur du lait maternel et la courbe de développement de l'enfant africain nourri au sein le prouve. Sa croissance somatique aussi bien que son développement psychomoteur sont supérieurs à ceux de l'enfant européen du moins dans les premiers mois de la vie. L'étude de cette courbe montre également que le kwashiorkor apparaît dans les semaines qui suivent le sevrage: 18 mois à Dakar, mais plus tôt à la Jamaïque ou en Amérique Centrale, où le sevrage est plus précoce.

D'où l'idée de prolonger l'allaitement maternel, de l'encourager en donnant une alimentation supplémentaire à la mère.

Certains travaux ont été faits dans ce domaine, en particulier au Congo Belge pares D^rs Lambrecht et Holeman.

Pour notre part, nous ne pensons pas que ce soit là une bonne solution au problème de l'alimentation de l'enfant.

Un essai fait dans un village du Sénégal avec la collaboration de l'O.R.A.N.A., de distribution de lait sec une fois par semaine, aux femmes allaitantes, ne nous a pas donné de résultats demonstratifs. Cet essai n'avait pas la rigueur d'une expérimentation, mais s'approchait des conditions qui seraient celles d'une distribution de masse.

Surtout deux autres arguments nous semblent importants. Le *premier* est l'étude de la courbe de poids obtenue au cours d'une enquête longitudinale, qui seule permet d'apprécier l'acroissement. Cette étude révèle que dès l'âge de 3 mois, l'accroissement de l'enfant africain est inférieur à celui de l'enfant européen, bien que le croisement des courbes de poids ne se produise qu'à 6 mois. La conclusion est que très tôt, le lait à lui seul ne suffit pas à assurer une croissance normale. Il faut lui ajouter d'autres aliments.

Le *deuxième argument* est d'ordre social. L'évolution naturelle accompagnant l'élévation du niveau de vie entraine le raccourcissement de la période d'allaitement. Vouloir la prolonger est aller à l'échec, et au cours de nos essais nous nous sommes heurtés à la mauvaise volonté du personnel africain chargé de cette action, qui jugeait la méthode rétrograde et se refusait à l'appliquer.

En conclusion, nous pensons que la tâche essentielle des services de P.M.I. n'est pas de chercher à prolonger l'allaitement maternel, pas plus d'ailleurs qu'à le raccourcir, mais bien plutôt de mettre au point et de favoriser la distribution d'un produit bon marché, d'une bonne valeur biologique, facile à utiliser et ne favorisant pas les diarrhées infantiles.

Dr. M. Parent (Congo Belge) — Nous avons obtenu 3 types de courbe de croissance chez les nourrissons noirs:

1) Une courbe qui coupe la courbe dite normale vers 3 à 5 ½ mois et reste en dessous de la normale, correspondant aux courbes habituelles moyennes observées;

2) Une courbe nettement en dessous de la normale dans des populations hyponourries (courbe type Vandergooten);

3) Une courbe dans une société minière où les femmes enceintes et allaitantes reçoivent un supplément alimentaire important

(viande, lait, etc.) et cette courbe est pratiquement la même que celle des enfants européens.

Nous concluons à l'intérêt de donner un supplément à la femme enceinte et allaitante pour l'amélioration de l'état de santé de l'enfant congolais.

PROF. A. LAMBRECHT (Belgique) — 1. En relation avec le *travail du D^r Bassir* et les remarques de M. Sénécal, voici notre opinion sur la supplémentation en protéines: nous sommes convaincus qu'en brousse (au Kwango, Congo Belge) il est préférable de donner le supplément non pas directement au jeune nourrisson, mais à la mère, et cela pour des raisons d'hygiène, de technique, de digestion et de bénéfice pour l'enfant.

2. *Réponse à M. Autret.*

a) Il n'y a pas de grande différence entre le besoin minimum en Ca déterminé par nous et les chiffres de 800 mg recommandés par le B. of N. Si on exprime les données en mg/kg/24 h, le poids moyen de l'Américain est très supérieur (± 60 kg) à celui de l'Africain du Kwango (± 43 kg). En règle générale, le besoin est couvert par le régime coutumier.

b) Pour ces expériences, nous avions $\frac{\text{Ca}}{\text{P}} = 0{,}68$, mais nous avons travaillé avec d'autres valeurs sans rencontrer de notables différences au point de vue de la rétention de Ca. Le rapport $\frac{\text{Ca}}{\text{P}}$ joue peu dans les conditions où nous opérons.

c) L'irradiation de la peau par le soleil intervient beaucoup dans le métabolisme du Ca. Il faut donc lutter pour maintenir ce facteur en continuant les habitudes coutumières (semi-nudité pour les adultes, nudité complète pour les jeunes enfants).

d) Les sujets que nous avons étudiés ici sont des sujets «normaux», c'est-à-dire des individus en bonne santé apparente, représentant l'indigène habituel du milieu coutumier du Kwango: ils sont de taille inférieure à la moyenne des sujets de race blanche. La quantité de Ca qu'ingèrent ces sujets paraît très probablement suffisante pour leur croissance *actuelle;* pour le moment, aucune donnée n'existe pour affirmer qu'une ingestion supérieure de Ca favoriserait la croissance ou serait nécessitée pour une augmentation de vitesse de croissance produite par une cause quelconque.

DR. M. DE SMET (Belgique) — Je voudrais demander quel est la pourcentage d'évolution vers la cirrhose dans le groupe des 71 % des hépatosplénomégalies autoentretenues.

Ensuite quelle est l'influence de la splénectomie dans ces cas? Nous avons efectué une soixantaine de splénectomies dans ces syndromes d'hépatosplénomégalie. Nous n'avons pas obtenu de régression d'une cirrhose constituée.

Ensuite, croient-ils dans l'existence d'une cirrhose splénogène dans ce groupe?

DR. O. BASSIR (U.K.) — I should like first of all to thank Dr. Adeniyi Jones (Medical Officer of Health of Lagos) and Dr. Ademola of the Infant Welfare Clinic in Lagos by whose courtesy my work was made possible.

A parallel experiment in which Dr. Ademola fed peanut flour to infants failed to show consistent increase in the body weight and growth rate of the subjects receiving the supplement. Probably the main cause of failure was the infection and diarrhoea which resulted from unhygienic conditions of the feeding of the flour.

Furthermore, the cheapest groundnut flour preparation which is at present available costs about 1/6 per child per weekly dose. This is much higher than the approximate cost of 60 per mother fed with soya flour, per week.

One more question I would like to pose is the evidence for the suggestion by an earlier speaker that the calcium requirement of babies depends upon their *rate* of growth. This is an important point in view of my reported observation of low daily intakes of Ca^{++} by breast fed Nigerian infants.

3RD SESSION: **Interrelationship between zymotic factors and malnutrition in diseases in the tropics**

Thursday, September 11th — 3:00 to 4:30 p.m. Room B

The meeting opened under the chairmanship of Dr. B. Maegraith.

The Rapporteur was Dr. J. C. Waterlow and the Secretary was Dr. Carlos dos Santos Reis.

Before the presentation of papers Dr. Maegraith asked the audience to keep one minute of silence as an hommage to Prof. A. Chandler, who died when he was coming to Lisbon to attend the Congresses.

Then the session was opened and the following papers were presented and discussed.

THE ROLE OF MALNUTRITION IN HELMINTHIC INFECTIONS

ASA C. CHANDLER [1]

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The effects of nutrition in parasitic infections have been the subject of several recent general discussions (Hunter, 1953; Chandler, 1953; Smith, 1955; Chandler, 1957). The purpose of the present paper is to call attention to the many and complex ways the diet of a host may influence the establishment, course and pathogenicity of parasitic infections. I shall refer only briefly to effects on specific immune reactions, since these have been treated extensively in the reviews mentioned above, as well as in Corkill (1950). Although

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[1] The Author did not attend the meeting.

I shall limit myself largely to helminthic infections, with which I am most familiar, much of what I have to say is applicable to other parasites as well.

In the past most experiments on effects of nutrition on parasitism have been concerned with the effects of gross factors such as deficiency of calories, protein, particular vitamins or minerals, or partial starvation, without reference to just how these deficiencies affect the parasites or their relations to the host, or how one deficiency might affect the entire metabolism of either the parasite or the host, or both. On the other hand, a deficiency may have very specific effects on a particular host-parasite combination, sometimes at only one point in the parasite's life cycle.

Constituents of a diet, or the diet as a whole, may affect the acquisition, course and pathogenicity of helminthic infections by influencing any of the following factors or processes: (1) ingestion of the infective stages of the parasites; (2) intestinal emptying time; (3) escape of infective larvae from eggs, cysts or sheaths; (4) penetration of the skin or mucous membranes by invading larvae; (5) provision of nutrients required by the parasite during various phases of its development in the host; (6) maintenance of satisfactory environmental conditions, suche as pH and oxidation-reduction potential, in the parasite's habitat; (7) replacement of lost blood and repair of damaged tissues; (8) control of hormone balance as it affects resistance and allergic reactions; and (9) specific immunological responses. In the present meager state of our knowledge of the physiology of helminths it is often impossible to determine just how dietary factors operate in favoring or repressing them. When dietary changes are extensive, it is probable that these may involve several different effects simultaneously, resulting in striking changes in the host-parasite relationship.

Such changes are sometimes spectacularly demonstrated by acquisition or loss of parasites when wild animals are fed artificial diets. Beaver (1937), for example, found that pigeons became refractory to infection with the trematode, *Echinostoma revolutum*, when fed on a laboratory diet; the pigeons did not lose weight and maintained excellent health, but showed changes in the character of the feces and were more restless. Oguri and Chu (1955) found that wild sea birds fed exclusively on squid were 100 % susceptible to *Parorchis*, a trematode inhabiting the cloaca, whereas domestic chickens and ducks on poultry feed were entirely refractory. When the ducks were placed on a squid diet, they too became susceptible. The only observed change in the cloacal environment was the absence of lactobacilli in the squid-fed birds. Another such example may be Whitten's (1954) observation that in New Zealand, although *Ascaris* is abundant in wild pigs, very few are harbored by domestic pigs, and none by those fed on whey.

An interesting example of acquisition of susceptibility is cited by Read (1958). The dwarf tapeworm, *Hymenolepis nana*, readily infects the gray squirrel, *Sciurus carolinensis*, when these are fed on dog biscuits in the laboratory, and it thrives even better in the squirrels than it does in its «natural» host, the mouse. Yet *H. nana* has never been reported from wild-caught *Sciurus*, could not be established in a wild population, and was lost from infected captive squirrels when these were provided with certain foods utilized by them in nature. Apparently the natural feeding habits of squirrels results in an intestinal environment unfavorable for *H. nana*. One cannot avoid the analogy of this to the effects of transplantation of Africans from the bush to an urban or suburban environment with accompanying changes in diet, graphically described by Elsdon-Dew (1954). In this case *Entamoeba histolytica*, although maintaining a similar incidence, changes from a relatively harmless lumen inhabitant to a tissue-invader causing severe amebiasis. Elsdon-Dew and Freedman (1952) also reported a great increase in *Trichuris* infections. Perhaps the spotty distribution of *Trichuris* in the United States and elsewhere is due in part to dietary factors, and not entirely to climatic and other outside environmental factors as usually supposed.

Let us briefly consider further some of the various ways, listed above, in which diet may influence parasitic infections. Ingestion of eggs of helminths with contaminated food or water, and of larval helminths with uncooked intermediate hosts need not be elaborated on, but a few special cases of ingestion should be mentioned in passing, e.g., *Fasciolopsis* with water chestnuts, *Fasciola* with water cress, guinea worm with *Cyclops*, in drinking water, and *Ascaris*, *Toxocara* and *Trichuris* with dirt-eating.

As to intestinal emptying time, Ackert and Nolf (1931) explained the effect of a deficiency of the vitamin B complex on the establishment of *Ascaridia* in chickens by the resulting partial paralysis of the gut. The importance of intestinal emptying time has also been demonstrated in other helminthic infections (Larsh, 1947; Read and Voge, 1954).

Little is yet known about the effect of diet on the liberation of parasites in the gut from eggs, cysts or sheaths. Recent work by Rogers and Fairbairn (personal communication) on hatching of *Ascaris* eggs, by Rogers and Sommerville (1957) on exsheathment of nematode larvae, and by Read (1955) and Rothman (1958) on excystment of tapeworm larvae has shown that all of these depend on a complicated interaction of factors involving host secretions. These factors are definitely concerned in host specificity, and also, according to Madsen and Whitlock, in genetically insusceptible individuals of a species (reported by Read, 1958). It seems probable that they might be affected by presence or absence, or amount, of dietary constituents as well.

The possible effect of diet on penetration of the skin and mucous membranes has received little consideration in the past. Recent work of Lewert and Lee and other workers (summarized in Lewert, 1958) has shown that penetration by certain helminth larvae involves enzymatic action against glucoproteins. The condition of these glucoproteins, and therefore their susceptibility to enzyme action of penetrating parasites, is affected by adrenal and sex hormones, by age, and also by diet. Lewert remarked that scorbutic animals have notably depleted, spongy, altered glucoprotein, and are known to be more susceptible to invasions, including invasion by *Entamoeba histolytica*. Possibly one of the ways in which vitamin A deficiency lowers resistance to parasites may be in connection with penetration. The lowering of resistance by vitamin A has been demonstrated in numerous host-parasit combinations, but always involving penetrating parasites.

If, as seems probable, diet does affect liberation or penetration of parasites, this could be a very potent factor. Schiller and Read, quoted by Read (1958), showed that although rodents are refractory to infection with *Echinococcus granulosus* when fed embryonated eggs, they are *not* refractory when material from hydatid cysts is injected intraperitoneally. Read also cited the work of Leonard and Leonard (1941), who showed that immunized rabbits refractory to infection with eggs of *Taenia pisiformis* were still susceptible when the onchospheres were injected into the mesenteric veins. As Read pointed out, the *Echinococcus* resistance might be due to absence of a necessary stimulus, and that of *Taenia* to inhibitory substances or reactions. However, both may involve enzyme action against glucoproteins, for this has been demonstrated in mucosal penetration by onchospheres of *Taenia saginata* (Silverman and Maneely, 1955), and in development of *T. taeniaeformis* in the liver of rats (Lewert and Lee, 1955).

The provision of nutrients necessary to permit normal growth, development and reproduction of parasites has been the subject of extensive research. A few comments of general nature must suffice here. That maintenance of parasites may be quite independent of establishment is exemplified by DeWitt's work (1956) on the effect of a deficient diet on schistosome infection in mice. Injury to the host lowered resistance so that many more worms established themselves, but the diet also failed to provide adequate nutritional support for the parasites, so they did not grow or develop normally. Apparent differences in symptomatology of schistosomiasis in different parts of Africa, to which Willard Wright called attention, might well be manifestations of obscure dietary differences that would not be apparent in a study of the general adequacy of the diets.

The nutrients, required by helminths may change at different stages in their life cycle. This is well demonstrated by the work of Goodchild (1958) on transplantation of schistosomes into the eyes of rats, and of Smyth (personal communication) on attempted cultivation of certain helminths *in vitro*.

The nutrient requirements of a parasite may be highly specific. Tapeworms, for instance, are strictly limited as to the kinds of carbohydrates they can utilize; these vary from species to species (see Read and Rothman, 1958), and the inclusion of such carbohydrates as lactose and fructose affects the utilization of others (see Read, Schiller and Phifer, 1958). There may be keen competition for nutrients between host and parasite, or between one parasite and another. When quickly-absorbed dextrose is substituted for starch in tapeworm-infected animals, the tapeworm suffers (Chandler, Read and Nicholas, 1950); when B_{12}-hungry *Dibothriocephalus latus* is situated far forward in the intestine, the host may suffer (Nyberg, 1958). When *Moniliformis dubius* and *Hymenolepis diminuta* are simultaneously present in the gut of a rat, the more rapidly growing *Hymenolepis* has its growth handicapped, apparently due to some scarce nutrient, as yet unidentified, being in inadequate supply (J. C. Holmes, unpublished).

Any dietary factor that influences secretion or utilization of bile might have unsuspected effects on parasites. Rothman and Read (1957) showed that the metabolism of glucose by *Hymenolepis diminuta in vitro* is inhibited by a low concentration of bile salts, yet Goodchild (personal communication) has found that *H. diminuta* will not survive *in vivo* at all in the *absence* of bile.

For repair of blood lost through the activity of blood-sucking worms, both protein and iron are needed. The effectiveness of supplementation of either one will obviously depend on the availability of the other. Here again there may be interaction between parasites, for the trypsin-inhibiting activity of *Ascaris* may cause an apparently adequate intake of protein to be inadequate, and there may be further loss of protein from anorexia, vomiting, impaired digestion and absorption, and diarrhea, one or all, induced by concomitantly present parasites. It should be emphasized that replacement of blood and development of specific immunity are quite independent processes. Antibody production seems to have a high priority for available protein and amino acids, even in cases of extreme emaciation (Balch, 1950). As I have pointed out elsewhere (Chandler, 1953*b*), it takes longer to develop immunity to worms that feed on blood than to those, like *Trichinella* and *Nippostrongylus*, that feed on local tissues; therefore there may be severe blood loss for several weeks before immunity develops, which even a good diet cannot replace. In fact, during this period a good protein diet may benefit the parasites more than the host, and account, in part, for the peculiar effect of supplemental feed for sheep, which results in more worms and more loss of blood in *Haemonchus* infections, but in enhanced resistance to the trichostrongylids that feed on local tissues (Whitlock, 1949).

Finally, I wish to call attention to the possibility of diet affecting the resistance of a host by influence on the endocrine system. Whitlock (1955) showed that lambs of ewes fed thiouracil during late pregnancy or early lactation were highly susceptible to trichostrongylidosis. He stated that anti-thyroid substances had been reported from various foodstuffs, and might play a part in susceptibility. On the other hand, Larsh (1950) found thiouracil not to alter susceptibility of mice to *Hymenolepis nana* infections.

Diet can affect parasites through action on the host's sex hormones. Growth of *Hymenolepis diminuta* is stunted in female rats on a diet that supports normal growth in adult males (Chandler, 1943), but this is rectified by addition of brewer's yeast. Later work by Addis (1946) and Beck (1952) showed marked effects of sex hormones on development of tapeworms in *both* host sexes which could be modified by diet, though more extensively in female than in male hosts.

ACTH and cortisone, by decreasing inflammatory response, may increase susceptibility to infection, and at the same time ameliorate allergic symptoms. Increased ACTH results from various stresses, including malnutrition (Selye, 1946). Read (1958) presented the intriguing idea that some of the diverse effects of malnutrition on parasites may be produced through pituitary-adreno-cortical stimulation.

From what I have said, it is obvious that there may be many different possible ways in which nutrition may affect parasites and parasitism, and that there is here a wide-open field for research. I should like to emphasize the need for much further laboratory research on experimental animals to determine the diverse and interacting effects of dietary constituents on particular parasites, and for field research to discover closer correlations than are now available between actual human diets and the prevalence and pathogenicity of particular parasites.

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ENDOCRINE GLAND ACTIVITY AND ZYMOTIC FACTORS IN THE VARIOUS FORMS OF PROTEIN MALNUTRITION

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Prof. A. C. Chandler was to have given the first paper in this Session on the «Interrelationship between zymotic factors and malnutrition in diseases in the tropics». As you have heard, he died in Rotterdam on his way to the Congress and many of us who had looked forward to meeting him were profoundly shocked when we had this news. I have felt for some years that I have known him through correspondence and from reading his papers and we must seek some consolation in the knowledge that he gave us a masterly account of his views on «Interrelations between nutrition and infectious disease in the tropics» in his Presidential Address to the American Society of Tropical Medicine and Hygiene in 1956 (Chandler, 1957). He opened this address with the remark that he doubted «if it would be possible in the whole realm of human knowledge to find a more perfect example of intertwining of cause and effect, with the production of a vicious circle, than that presented by malnutrition and infectious disease [...] These are two specters which haunt the great majority of the inhabitants, from conception to the grave, in the so-called tropics [...] The backwardness of many tropical peoples [...] is the result of injury by these two specters, rather than of any climatic effects or innate inferiority».

Consideration of this important subject must necessarily be seriously hampered by the tragic loss of Dr. Chandler and I must therefore, since we do not have the paper he was to have given on

«The role of malnutrition in helminthic infections», turn to the abstract of it in order properly to introduce the subject. He says:

> The course of a parasitic disease depends on numerous factors, including susceptibility of the host to invasion; provision of satisfactory physical and chemical environmental conditions for the parasites; and ability to develop chemical or physical alterations which will interfere with their invasion, growth, development, and reproduction.

and he concludes:

> In appraising the interrelations of malnutrition and parasitism it is, of course, necessary to consider also the additive effects of parasitism on malnutrition through anorexia, vomiting, diarrhea, impaired liver function, fever, neutralization of digestive enzymes, absorption of vitamins, loss of blood, etc.

I suspect that he intended to emphasise the importance of malnutrition in resistance to infection but he adds «malnutrition may serve as a stress that increases adrenocortical activity, thereby decreasing inflammatory responses and consequent lowering of resistance».

This is my cue for considering endocrine gland activity in relation to the zymotic factors in malnutrition.

I have examined in previous papers (see Platt, 1958*a*) various aspects of this interplay between malnutrition and zymotic factors in disease, but I should for clarity here repeat that zymotic disease strictly speaking means infectious disease due to fungi; the term «zymotic factors» is a convenient one to include all living organisms that produce disease, i.e. it includes all infections and infestations.

Three ways in which the subject of the nutritional status of the host in relation to zymotic factors can be examined are:

a) Its effect on the susceptibility and resistance to, and the course of the infection — this is the approach most commonly adopted;

b) Its relationship to nutritional requirements of the metabolism of the infecting agent — a viewpoint which has been profitable in some chemotherapeutic developments;

c) The contribution of the zymotic factors to the development of malnutrition in the host.

There is no time now to discuss even these three aspects of the subject in detail. I would like, however, to draw your attention to a phenomenon which has been known for many years (see Platt, 1957) — the so-called toxic destruction of protein. There may be, after fever accompanying bacterial infections, a loss of as much as 2.5-4.5 kg of muscle tissue together with a loss as great as 7.5 kg in body weight. In one disease found in the tropics—Rocky Mountain spotted fever — sometimes as much as 10 g of dietary protein per kilogramme body weight daily is needed to produce positive nitrogen balance (Harrell, Wolff, Venning & Reinhart, 1946). These losses, together with impaired appetite, deranged function of tissues, and increased metabolic activity during fevers, must inevitably result in malnutrition. Some losses in the other constituents of the body may be expected to occur. The complex nature of these losses in injury is well recognised in surgical practice. In medicine we should not be behind in recognising not only these losses of nitrogen from the body, but also possibly of other nutrients, and that to counteract them we must give enough of an appropriately balanced diet.

Though we do not yet fully understand the mechanism of these losses to the body caused by infection and injury, they are no doubt related to the effects of stresses on endocrine gland activity. I want now to present two examples of interrelationships which involve malnutrition, endocrine disturbance and zymotic factors in disease; such interrelationships may include a combination of two or all three of these factors [1].

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[1] Attention was drawn here to an exhibit of the Human Nutrition Research Unit of the Medical Research Council of the United Kingdom which included more than fifty photographs, microphotographs and diagrams illustrating the production in the experimental animal of syndromes resembling the two extremes of types of protein malnutrition, i.e. those recognised clinically as marasmus and kwashiorkor. (Many of the items in the exhibition will be found in Platt, 1958*a*, and Platt, 1958*b*.) These two forms were produced by feeding a low-protein diet (LP) and the same amount of low-protein diet with additional carbohydrate (LP+CH). Fig. 1 in Platt, 1958*b*, in particular summarises a few of the basic data on the observations on the piglet.

TRANSVERSE TRABECULAE IN BONE

Bands of dense tissue in bone have been recognised for many decades, even before the introduction of radiology, but in the early 1920's thin transverse lines in radiographs of the growing ends of the long bones of children were recognised (Harris, 1924, 1926). These were generally related to some ilness such as measles or whooping cough and they have also been associated with endocrine disturbances, notably episodes of uncompensated diabetes in children. We have recently produced arrested growth lines, single and multiple, in the bones of pigs on low protein diets (Stewart & Platt, 1958).

LESIONS OF THE PANCREATIC ENDOCRINE TISSUE AND FATTY LIVER

Fatty liver is well known to occur in association with some acute infections. It is also a recognised feature of the pathology of protein malnutrition. Recently Gillman & Gilbert (1958) have produced fatty liver of the type seen in protein malnutrition in baboons by producing a relative or absolute insufficiency of insulin. The main factor of this infection-malnutrition-endocrine interrelationship might well be suspected to be an alteration in the endocrine development of the pancreas, i.e. the islets of Langerhans. In the piglet on a low-protein high-carbohydrate diet, a marked reduction in the size of the islet cells has been found, the amount of cytoplasm in the islet cells being reduced (Heard, Platt & Stewart, 1958); and the proportion of the α to β cells is altered (see Platt, 1958*b*, Plate H). Moreover homeostasis with respect to sugar metabolism of the body is affected; the blood sugar, after administration of insulin, shows an exaggerated fall and a delay in return to normal values as compared with the response of the normally-fed animal.

CAUSE OF DEATH IN ACUTE FULMINATING PROTEIN MALNUTRITION

Mention has been made (Platt, 1958*b*) of the fact that animals with experimentally-produced protein malnutrition and some poorly nourished Gambian infants die a similar death, which is suspected to be caused by failure of function of parts of the central nervous system. This may be a combination of two factors: (*a*) failure of protein anabolism in the neurones, and (*b*) breakdown in the energy supply to the central nervous system. We have obtained evidence of pathological changes in the Nissl granules of neurones of parts of the central nervous system and it is believed that these granules are a site of protein synthesis. It is generally held that the brain, of all the organs in the body, is unusually dependent on glucose as its source of energy and that it has only a very limited ability to oxyidise substances other than glucose when glucose is lacking.

It would seem that there is sufficient evidence to make it worth while to investigate the possibility that these two phenomena are the immediate cause of death in the acute fulminating form of protein malnutrition. It is noteworthy in this connection that Waterlow (*Proc. 6th Int. Congr. Trop. Med. and Malar.*, **6**: 158-168, 1959) reports in his acute cases a 20 per cent mortality for which he cannot ascribe a cause. The observations reported above also suggest that administration of insulin and glucose, along with the measures already employed for treating protein malnutrition, might reduce mortality in these acute cases.

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CIRRHOSIS AND DISSEMINATED CALCIFICATION OF THE PANCREAS IN PATIENTS WITH MALNUTRITION

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18 Indonesian patients suffering from disseminated calcification of the pancreas were observed at Jogjakarta (Java) from 1953 till 1957. This diagnosis was made by roentgen-examination. As is well known, such a diffuse pancreatic calcification only occurs in a markedly fibrotic pancreas. Fibrosis of the pancreas may or may not be followed by calcification.

A similar clinical picture as presented by these patients, but without calcification of the pancreas, was observed in 27 other patients. It was assumed that these patients were suffering from fibrosis of the pancreas without calcification.

A first report about 13 patients was given in 1954 (1). This paper means to discuss these 45 patients with pancreatic fibrosis.

CLINICAL DATA

13 were females, 32 males. It is apparent from Table 1 that 19 patients were less than 21 years old and 34 less than 31 years.

TABLE 1 — *Age of 45 patients with pancreatic fibrosis*

12–15	16–20	21–25	26–30	31–35	36–40	41–45
7	12	8	7	4	6	1

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 287-292, 1959

In 43 patients *diabetes* was the reason for admission. They presented the classical symptoms of this disease: polydipsia, polyuria, polyphagia and emaciation. Weakness and fatigue were common complaints. Many patients were already in an advanced stage of the disease because they sought medical treatment not before their vision became seriously impaired as a result of bilateral cataract.

The diabetes was often severe. As shown in Table 2, in 17 patients the fasting blood sugar level before treatment was between 200 mg and 300 mg per 100 ml, in 16 patients between 300 mg and 400 mg per 100 ml and in 2 patients this value exceeded 400 mg per 100 ml.

TABLE 2 — *Fasting blood sugar level (in milligrammes per cent) in 43 patients with pancreatic cirrhosis and diabetes*

100-150	150-200	200-300	300-400	More than 400
2	6	17	16	2

It is therefore not surprising that all patients were more or less emaciated; in none the body-weight exceeded 40 kg and often the weight was less than 30 kg. They showed no oedema, obviously as a result of polyuria.

The diabetes was difficult to control, insulin resistance was often noticed. While in hospital, the patients received a diet of 1,500 to 2,000 calories, containing 100 g to 150 g carbohydrates. With this diet, all patients needed insulin. Only in 10 patients the insulin requirement was less than 40 units to obtain satisfactory blood sugar levels. 22 patients received from 40 to 100 units and 8 patients needed 100 to 200 units a day; nevertheless, in these 30 patients blood sugar levels often remained exceedingly high. From 3 patients accurate data are not available.

In all patients ketosis was conspicuous by its absence. When they left the hospital, insulin treatment had to be stopped; however, the withdrawal of insulin did not result in ketosis.

Some diabetic complications were often seen: severe pyorrhoea, gingivitis, loosened teeth. Bilateral cataract was present in 15 patients, some of them being practically blind.

X-ray examination showed disseminated pancreatic calcification in 18 patients. In 2 patients the calcification was still in the initial stage, being restricted to the head of the pancreas; in all other cases the calcifications were scattered throughout the whole pancreatic area, but most intense in the region of the head of this organ. In a few patients the X-ray gave the impression of an atrophied, almost totally calcified pancreas, crossing the spinal column as a narrow ribbon.

On clinical grounds no differentiation could be made between the patients with and those without calcification.

In such extensive pancreatic lesions severe disturbances in the external secretory pancreatic function might be anticipated. However, this was not the case. None of the patients complained of diarrhoea and the aspect of their stools was normal. Microscopically, with Sudan III more or less neutral fat could only be found in the stools of 10 patients. Creatorrhoea was not observed. Examination of duodenal juice was carried out in 20 patients: a low lipase content was a constant finding, but the concentration of trypsin and amylase was normal in nearly all patients.

Though no quantitative fat balance studies could be carried out, the conclusion is warranted that the external secretory function of the pancreas was not markedly disturbed.

In 2 patients with disseminated calcification of the pancreas no diabetes existed. In the first the condition was accidentaly discovered during fluoroscopy of the thorax. The other one complained of pain, its localization and nature were characteristic of «pancreatic pain»; the pancreas was markedly enlarged, the head could easily be felt as a firm swelling. It was assumed that some calcification, by pressure on pancreatic ducts, inhibited the flow of pancreatic juice and for that reason became the cause of pancreatitis.

The absence of steatorrhoea certainly accounts for the fact that this severe pancreatic disease was not recognized at an earlier date, as many similar cases were already seen before 1953. At autopsy of such a case (1939) an atrophic, firm pancreas was found, microscopical examination showed that nearly all parenchyma had disappeared and been replaced by fibrous and adipose tissue.

PATHOGENESIS

According to the literature, fibrosis of the pancreas is the end result of a chronic, relapsing pancreatitis. This disease often runs a protracted course with acute exacerbations of pancreatic inflammation. Ultimately the acinar tissues atrophy, marked inter- and intralobular proliferation of connective tissue develops, which leads to severe interstitial fibrosis in which calcium salts may be precipitated (2-10).

However, the clinical picture of our patients did not tally with a diagnosis of chronic pancreatitis. This is obvious, if the symptomatology in patients with chronic pancreatitis is compared with that in our patients (Table 3).

TABLE 3 — *Symptomatology in patients with chronic pancreatitis and in our patients*

	Literature	Our patients
Age	Middle-aged	Young adults
Sex	Chiefly males	Males and females
Attacks of pancreatitis in history	Often present	Never
Alcoholism.	Often	Never
Pain	Principal symptom	Absent in all but one patient
Steatorrhoea	In 25 per cent	Principal symptom in none
Diabetes mellitus	In 25–40 per cent	Principal symptom in 43 patients

If chronic pancreatitis can be ruled out, we must look for another cause of this pancreatic disease.

We must assume that our patients were suffering from a severe, degenerative afection of the pancreas parenchyma, leading to atrophy of the acinar tissue with fibrous tissue gradually replacing the parenchyma; in this process the islands of Langerhans became eventually involved. In our opinion this disease is caused by protein malnutrition. The following reasons support this view:

1. All patients belonged to the very poor classes of the population; their diet was very low in animal protein and inadequate as regards total calories. In the same group, cirrhosis of the liver is very common. (In our patients a clinical diagnosis of cirrhosis of the liver could be made in none, though a disturbed hepatic function could be demonstrated in a few.)

2. 16 patients showed enlargement of the parotid glands; in some patients this swelling was very conspicuous. This condition is considered to be related to protein malnutrition.

3. In 15 patients changes of the hair of the scalp were noticed: often it was sparse, could easily be removed by pulling or combing and had changed its colour from black to brownish-black; often a reddish hue was noticed. Such changes are well known in kwashiorkor (11).

4. In kwashiorkor striking changes are found in the pancreas. Many authors assume that the major damage in kwashiorkor is not to the liver but to the pancreas. Microscopic examination shows atrophy of the acinar cells followed by proliferation of fibrous tissue in and around the lobules (12-14).

It seems therefore that the severe pancreatic lesion in our patients is a result of protein malnutrition. Kwashiorkor in children is often seen in Indonesia (15). Our patients belong to the same group of the population, in which kwashiorkor in toddlers often occurs. It is not astonishing that the pancreas, which suffers so much from protein deficiency in infancy, shows extensive lesions in adults which have been exposed to an inadequate diet ever since childhood.

According to Trowell *et al.* (l. c.), a definite sclerosis of the pancreas is uncommon in children suffering from kwashiorkor but quite frequently encountered in adults. However, in his adult patients with protein malnutrition diabetes did not exist. This marked difference between his African and our Indonesian patients remains unexplained.

Hugh Jones (16) describes similar cases of diabetes from Jamaica and adds that this type of diabetes is reported from Ibadan (Nigeria) and Malaya. This author suggests that this peculiar type of diabetes may be generally common in tropical countries. However, in these cases no examination of the pancreas is mentioned.

In our opinion, emphasis should not be placed upon a special type of diabetes in tropical countries, but on the occurrence of pancreatic cirrhosis with their sequelae in tropical regions as a result of malnutrition.

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THE MECHANISM OF MALARIAL HEPATOMEGALY AND ITS RELATIONSHIP WITH HEPATIC FIBROSIS

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A number of surveys, such as those of McGregor and Smith (1952), Black (1954) and Walters (1958), have shown in holoendemic malarial areas a close parallel between the incidence of splenomegaly and that of hepatomegaly during the first few years of life. Bruce Chwatt (1945) has demonstrated that, with reference to English children, the livers of African children dying in Lagos were heavier until the age of six years, after which age their mean weight was exceeded by that of the livers of European children of the same age. Black (1954) has reported that adult Polynesians, transported from the non-malarious Wallis Islands into a highly malarious environment in the New Hebrides, rapidly developed hepato-splenomegaly unless protected from malaria. Furthermore, McGregor and his associates (1956), by protecting with a prophylactic dose of chloroquine given from birth every second child born in a Gambian village, have carried out a critical experiment which showed that at 3-3 ½ years of age the protected children — excepting one suffering from congenital syphilis — had livers of normal size, and impalpable spleens, while the control children all showed marked hepato-splenomegaly.

It has long been recognised that Laennec's cirrhosis has a far higher incidence in the tropics than in non-malarious temperate countries and the relationship of this condition with malarial infection has for 50 years been a matter of controversy. Thus while Hance (1938) has stated a case for the existence of a true malarial cirrhosis, Vint (1931) has denied the existence of such an entity.

Light has been thrown on the mechanism of hepatomegaly in malarial children by comparing in McGregor's Gambian series the histology of the liver, as displayed in punch biopsy specimens, of the protected group with that of the unprotected group which had been subject to heavy and repeated malarial infections. The outstanding differences shown by the malarial livers comprised:

a) Sinusoidal dilatation.
b) Hypertrophy of Küpffer cells.
c) Cellular infiltration of sinusoids.
d) Excessive fibrous tissue in portal tracts and in immediate periportal zone.
e) Malarial pigmentation.

Both groups of children, which had been reared in the same environment, were well-nourished, and the histological differences are attributable only to malarial. From this experiment it would appear that the transient hepatomegaly of childhood, which subsides on the attainment of premunity, is a dynamic process in which the main contributing factor is sinusoidal dilatation. However, these studies fail to explain the tendency towards the development of Laennec's cirrhosis in later life, since the portal and periportal fibrosis noted in the malarial group was minimal. Waterlow and I (Walters and Waterlow, 1954), in a large series of Gambian children ranging from infancy to adolescence, traced the development of the Laennec type of diffuse cirrhosis from an early portal and periportal fibrosis of infancy. This sequence of events was often well-advanced by the third or fourth year. The majority of our specimens showed minor evidence of nutritional parenchymal damage in piecemeal necrosis of liver cells, while an acidophil appearance of some surviving cells appeared to indicate degeneration due to loss of desoxyribose nucleic acid. We postulated that the Gambian form of juvenile hepatic fibrosis had a dual aetiology, in which a heavy and repeated malarial infection irritated the stroma by a bombardment with macro-molecules of malarial pigment, thus making it over-reactive to an otherwise insignificant degree of nutritional damage to the parenchymal cells. The subsequent study of McGregor's two groups appears to support this hypothesis.

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PAROTIDOSES DES CIRRHOSES ÉTHYLIQUES EN FRANCE ET PAROTIDOSES DE MALNUTRITION EN GUINÉE FRANÇAISE

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Nous avons attiré en 1954 l'attention sur les grosses parotides dans les cirrhoses alcooliques (1) et notre élève Catanzano a consacré à ce sujet sa thèse en 1956 (2).

Sous la direction de Raoult et la nôtre, Thomas, en 1955 (3), étudiait la parotidose de dénutrition, avec une référence plus particulière aux parotidoses observées en A.O.F., grâce au matériel très important confié par Raoult.

Nous nous sommes depuis posé la question de savoir si, comme il le semblait, les modifications histologiques et histochimiques des parotides dans les cirrhoses éthyliques et les malnutritions tropicales étaient absolument identiques. (C'est pourquoi nous avons tenu à examiner des parotides fixées et colorées dans des conditions exactement semblables à celles observées en 1954.

TECHNIQUE DE FIXATION ET DE COLORATION

La fixation est faite dans le liquide de Helly fraichement préparé (9 volumes de Zenker pour 1 volume de formol). Le prélèvement doit être fait moins de deux heures après la mort ou effectué par biopsie. Signalons au passage que nos biopsies ne nous ont jamais donné aucun accident, en particulier pas de fistules. Les fragments d'une épaisseur maximum de 5 mm séjournent six heures dans le

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fixateur et sont ensuite lavés six heures. Puis, on les passe dans l'alcool.

Les colorations pratiquées ont été: l'hémalun-erythrosine-safran, la méthode trichrome lente de Masson, la méthode de Hotchkiss-Mac Manus, la réaction d'Unna-Pappenheim, le mucicarmin.

ASPECTS HISTOLOGIQUES ET HISTOCHIMIQUES DES PAROTIDES DES CIRRHOSES ÉTHYLIQUES

Nos prélèvements ont été faits le matin à jeun dans le cas de biopsie, de façon à éviter des différences dues à la digestion.

L'*aspect aquipare* est le plus frappant. On se trouve devant un aspect analogue à celui d'une glande muqueuse ou de la glande lacrymale, alors que les acini normaux sont sombres. Les acini sont ici faits d'éléments à cytoplasme clair, spumeux; les noyaux sont rétractés, rejetés à la base. La réaction de Mac Manus ne colore aucun grain de sécrétion. La réaction d'Unna-Pappenheim ne montre pas d'acide ribo-nucléique. Il n'y a pas d'inclusions lipidiques, ni glycogéniques. La réaction de muci-carmin est négative. Devant cet aspect on songe aux «cellules aquipares», terme par lequel Renaut désigne les cellules des glandes lacrymales.

La *suractivité caricaturale* est le fait de plages voisines. Elle donne à l'aspect aquipare sa signification de phase terminale d'épuisement par fonctionnement exagéré. Chacun des stades sécrétoires s'exagère et il existe une alternance nettement tranchée entre les divers territoires glandulaires.

Au stade «zymogène», les grains de sécrétion sont très nombreux, grossis et accumulés dans toute la cellule.

Au stade «séreux», il y a engorgement et structure brouillée des acini.

Enfin, le stade «aquipare» traduit l'épuisement cellulaire et la période de repos.

Ces aspects diffèrent notablement de celui de la parotide normale, où seul figure le stade granuleux, alors que dans les glandes muqueuses on voit seulement la deuxième période du cycle avec un mucus parfait remplissant la cellule.

Les cellules sont hypertrophiées, augmentant le volume des acini et effaçant sa lumière.

De plus, on voit des foyers d'adénomatose au voisinage de passages de Boll et même une adénomatose franche et diffuse.

Les cellules sont alors petites, avec un noyau arrondi. En dehors des plages d'adénomatose, la modicité de la substance ergastoplasmique, colorée par la méthode d'Unna-Pappenheim, s'oppose à la surabondance des mucopolysaccharides, révélée par l'oxydation periodique dans les stades zymogènes et séreux. Il y aurait donc utilisation très rapide de l'acide ribonucléique. On comprend alors l'aspect clair des cellules épuisées en regard de l'activité amylolytique considérable de la salive (6.000 à 8.000 unités Wohlgemuth au lieu de 2.000 à 4.000).

Au stade «aquipare», le trichome de Masson et la réaction à l'acide périodique-Schiff montrent un épaississement accentué des basales acineuses. Ce phénomène dépend de modifications physicochimiques des glycoprotéines de la vitrée.

Par ailleurs la sclérose est généralement peu accusée et se limite à quelques épaississements de cloisons interlobulaires.

Le tissu adipeux n'est pas augmenté, sauf chez les obèses cirrhotiques. Il n'existe pas de dépôt pigmentaire ferrique.

En conclusion, on voit donc qu'il s'agit d'une parotidose caractérisée par l'hyperactivité, l'hypertrophie et l'hyperplasie des éléments cellulaires. *L'individualisation d'un stade aquipare d'épuisement, l'exagération des phases visibles de la sécrétion dans les stades granuleux et séreux préalables, confèrent aux parotides du cirrhotique alcoolique une originalité remarquable.*

ASPECTS HISTOLOGIQUES ET HISTOCHIMIQUES DES PAROTIDES DE MALNUTRITION EN GUINÉE

Voici un exemple caractéristique de parotidose par malnutrition:

Toure A. ..., 7 ans, indigent, entre à l'hôpital de Ballay à Konakry, dans un tableau d'anasarque.

Ce jeune garçon présente un très mauvais état général, avec une émaciation du visage et des mains, qui contraste avec le gonflement œdémateux du tronc et des membres inférieurs. Il existe en effet des

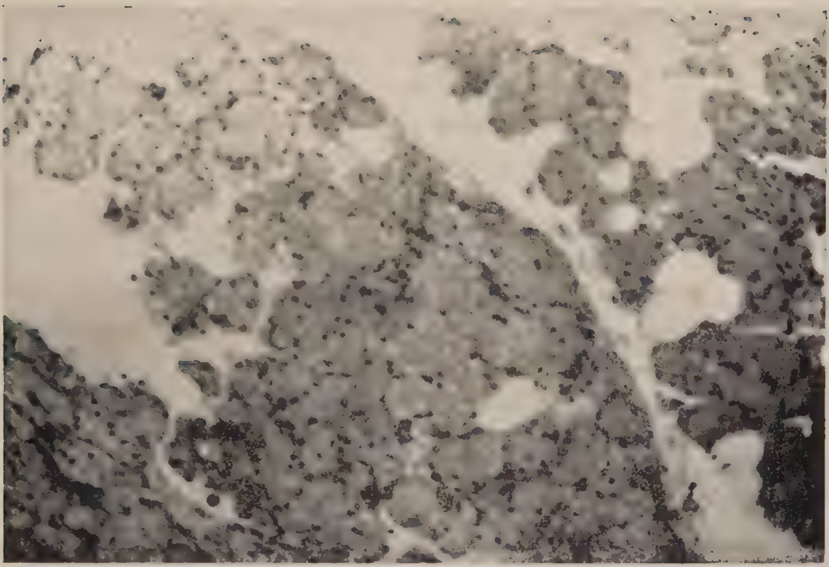

Fig. 1 — *Parotide de cirrhose éthylique.* Deux territoires contigus dont l'un, en bas, est au stade séreux et dont l'autre, en haut et à gauche, est au stade aquipare. On notera dans ce dernier la clarté quasi végétale des cellules

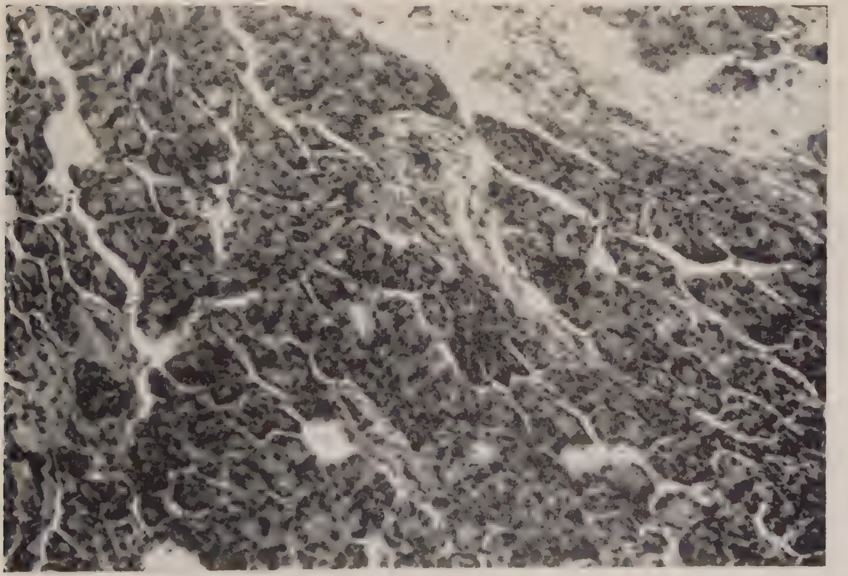

Fig. 2 — *Parotide dans un syndrome de dénutrition (Guinée).* Même technique de coloration. Mêmes constantes optiques et photographiques que ci-dessus On notera comparativement la petite taille des cellules, la grande richesse nucléaire du parenchyme et l'importance des travées qui le pénètrent

œdèmes très importants, mous, prenant le godet, avec par endroits des ulcérations atones à l'emporte-pièce. L'enfant est très fatigué, ses conjonctives sont pâles, et il répond à peine aux questions. Il présente une diarrhée chronique. On note de grosses parotides et un gros foie lisse, indolent, débordant de 4 travers de doigt le rebord costal. La rate est grosse, débordant de 3 travers de doigt. Il n'y a pas d'albumine dans les urines. L'examen du sang montre une anémie à 2.100.000 G.R. et 12.800 leucocytes avec une formule normale. Il n'existe pas de parasites ni dans le sang, ni dans les selles. Un traitement comportant méthionine, extraits hépatiques, calcium, vitamine C, delta-hydro-cortisone, tonicardiaques, etc., n'empêche pas la mort, qui survient après 20 jours d'hospitalisation.

EXAMENS ANATOMOPATHOLOGIQUES.

Le foie se montre divisé par de très larges bandes de sclérose, riche en fibroblastes et parsemée de néocanalicules biliaires. La structure normale n'est plus reconnaissable et les travées conjonctives cernent des territoires où les cellules hépatiques sont presque toutes frappées d'une stéatose à grosses flaques ou en dégénérescence homogène atrophique. Il n'existe aucun pigment, en particulier pas de pigment malarique.

Les parotides montrent une sclérose périlobulaire et péricanalaire. De très vastes plages sont très hématéiphiles par l'adénomatose très marquée.

Quelques acini sont au stade zymogène et d'autres au stade «aquipare». Au Mac Manus, les cellules acineuses sont bourrées de grains de sécrétion avec noyaux petits, refoulés vers la basale.

Une différence paraît, immédiatement nette. La sclérose, fort discrète dans les parotides des éthyliques, est dans celles des sujets morts en état de malnutrition beaucoup plus importante. Inter et intralobulaire, elle découpe les plages microscopiques. Peu inflammatoire, elle ne comporte pas de dépôt ferrique, contrairement à ce qu'ont observé Gillman, Gilbert et Gillman chez les Bantous (4).

Cette richesse en tissu conjonctif dense se trouve même chez le jeune, en Afrique Noire. Elle n'est pas particulière aux parotides, mais intéresse aussi le foie, le pancréas. On serait tenté de voir là une conséquence d'une nourriture déséquilibrée de la mère pendant la gestation, à une période où les besoins en protides sont considérables.

Dans l'étude de Thomas, qui a utilisé les résultats obtenus par Raoult en A.O.F. et plus particulièrement au centre de Popenguine, les sujets examinés ont été des enfants de 0 à 12 ans. On peut noter que le conjonctif peut être normal ou au contraire augmenté.

Quant au parenchyme, si nous retrouvons dans ces parotides des aspects «aquipares» d'épuisement, avec pauvreté de l'ergastoplasme, des signes de suractivité par la richesse en grains de sécrétion d'autres plages, l'épaississement des basales acineuses, un fait nous paraît différent de ce qu'on constate dans les parotides des alcooliques, c'est l'importance de l'adénomatose.

En bref, la parotidose de la malnutrition en Guinée Française nous semble plus particulièrement caractérisée par l'existence d'une sclérose et d'une adénomatose importante.

CONCLUSIONS

Les parotidoses des cirrhoses alcooliques sont caractérisées par l'aspect aquipare d'épuisement par hyperfonctionnement, l'exagération des phases visibles de la sécrétion dans les stades granuleux et séreux préalables, la discrétion de la sclérose et de l'adénomatose.

Les parotidoses des états de malnutrition en Guinée comportent par contre une notable sclérose, une adénomatose importante et l'aspect aquipare est moins important.

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LES PROTÉINES SÉRIQUES DES PAPOUS DE LA NOUVELLE GUINÉE NÉERLANDAISE EN RAPPORT AVEC LE RÉGIME, LES MALADIES INFECTIEUSES ET LA RACE

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INTRODUCTION

Depuis la guerre mondiale 1940-45, plusieurs publications ont paru concernant la nutrition et les conditions nutritionelles des habitants de la Nouvelle Guinée Néerlandaise (6, 9, 10, 11). Tous les auteurs font mention d'une situation nutritionnelle insuffisante dans plusieurs régions du pays.

Bliek, en collaboration avec Bakker et Duyken (2), a été le premier à étudier les protéines sériques des Papous. Il examina un groupe de Kapaukus, originaires des Wisselmeren, où ils vivaient à une altitude de 1.700 m environ. Les anomalies principales étaient une diminution nette de l'albumine et une augmentation des gammaglobulines. La valeur des protéines totales était inchangée comparativement aux valeurs européens. Des résultats semblables on été obtenus par d'autres auteurs (1, 3, 13).

Les causes des changements mentionnés ci dessus sont:

1. Un menu déficient (4, 5, 14).
2. Des maladies infectieuses (12, 15, 8).
3. La race (4, 5, 7, 16).

En ce qui concerne le régime et les maladies infectieuses, nous citerons les travaux de Klerks (en Indonésie), Holemans, Close,

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Demayer, etc., au Congo Belge, Gopalan, etc., aux Indes. Quant à la race, les résultats obtenus par Reinhold c.s. font présumer un facteur racial. Close et Jelliffe n'ont pas trouvé une telle influence raciale.

Pendant notre séjour dans la Nouvelle Guinée Néerlandaise, on nous a fourni l'occasion de visiter quatre régions:

1. *Biak,* où les habitants vivent à une altitude de 300 m au-dessus du niveau de la mer. Le paludisme antérieurement hyperendémique est combattu avec succès; le pian a été exterminé. Des aliments, surtout d'origine végétale, forment la base de l'alimentation. Les aliments d'origine animale sont rares.
2. *Nubuai,* un village à la côte septentrionale du continent. On n'y mange que du sagou. Quelques crustacés sont pêchés dans les bouches des rivières. Le paludisme y sévit sous forme mesoendémique. Le pian, comme à Biak, a disparu.
3. Le *Mappia* est une région des montagnes centrales. Il n'y a pas de paludisme, et plus de pian. L'altitude moyenne, où les habitants vivent dans des circonstances difficiles (la base de leur menu est la patate douce), est à peu près 2.000 m. Les aliments d'origine animale: grenouilles, rats, serpents, etc., n'entrent pas régulièrement dans la composition des repas.
4. *Sorong.* Les ouvriers d'une société pétrolière y ont été examinés. Leur menu est varié et probablement adéquate. Grâce aux soins médicaux intensifs, l'importance du paludisme est réduite; le pian n'y existe plus.

LES PROTÉINES SÉRIQUES DES PAPOUS DANS LES DIFFÉRENTES RÉGIONS

Le tableau 1 vous montre: 1) des taux des protéines totales qui sont du même ordre que ceux trouvés habituellement chez la race blanche; 2) le rapport albumine/globulines est inférieur. La seule exception est formée par les données obtenues à Sorong. Les analyses du sérum d'un groupe de filles et de garçons travaillant à l'hôpital de Biak s'approchent de celles des Néerlandais de Biak.

TABLEAU 1 — *Les taux des protéines sériques des Papous des régions différentes*

Région	Nombre examiné	Prot. tot. g %	Alb. g %	Alpha g %	Beta g %	Gamma glob. g %	Rapport A/G
Biak	315	7,13	3,23	0,92	0,72	2,26	0,83
Nubuai	298	6,76	3,15	1,01	0,72	1,88	0,87
Mappia.	156	6,26	2,90	0,95	0,71	1,70	0,86
Sorong.	166	6,99	3,62	1,01	0,77	1,59	1,07
Biak ville.	31	7,20	4,02	0,89	0,69	1,60	1,26
Résidents néerland.	18	7,08	4,35	0,80	0,69	1,24	1,59

1. L'INFLUENCE ALIMENTAIRE.

Prenant l'agrandissement du foie comme critère de la condition nutritionnelle, nous voyons qu'il n'existe pas ou presque pas de corrélation entre la grosseur du foie et les teneurs en protéines sériques, ni entre les différents constituants.

Comparant les analyses sériques de ceux avec et sans agrandissement du foie et de la rate, nous remarquons une diminution des taux albuminiques combinée avec une augmentation significative des taux gamma globuliniques. Il n'y a pas de doute que l'influence de la malaria soit plus importante quand les conditions nutritionnelles laissent à désirer.

2. L'INFLUENCE DES INFECTIONS.

Nous venons de faire remarquer que l'influence de la fièvre paludienne est nette. Les sujets présentant un agrandissement de la rate montrent un Rapport A/G moins favorable que les autres avec une rate impalpable. Nous avons l'impression que l'influence de la malaria est proportionnelle à son importance. L'élévation des valeurs des protéines totales est probablement due à l'augmentation des taux gamma globuliniques.

Les changements que nous venons de mentionner sont encore plus remarquables quand nous mettons en cause le pian. Surtout les lésions chroniques sont d'une grande importance.

Les infections des parasites intestinales causent principalement une baisse des taux des protéines totales, qui d'ailleurs n'est pas très importante.

3. LA RACE.

À notre avis, les déficiences nutritionnelles et les infections jouent les rôles les plus importants dans l'origine des changements dans le spectre protéiniques. Considérant le tableau 2, nous constatons que les spectres des nouveaux nés de Biak, selon qu'ils habitent l'île de Biak ou à Sorong, ne sont pas identiques à ceux des nouveaux nés de race blanche. D'autre part, les analyses des Indonésiens et des Chinois sont du même ordre que les analyses effectuées sur des sujets européens. Ces derniers sont en général mieux nourris et vivent sous des conditions plus hygiéniques. Il nous semble que le facteur racial est encore très problématique.

TABLEAU 2 — *Les taux des protéines sériques des nouveaux nés*

Région	Nombre examiné	Prot. tot. g %	Alb. g %	Alpha g%	Beta g %	Gamma glob. g %	Rapport A/G
Biak	24	5,63	3,25	0,72	0,49	1,17	1,36
Sorong.	7	6,49	3,71	0,90	0,59	1,29	1.33
Sorong (Indonésiens)	9	5,59	3,40	0,62	0,39	1,19	1,55
Biak (Chinois) . . .	2	5,58	3,40	0.61	0,39	1,17	1,57

RÉSUMÉ

D'après nos résultats, l'influence des maladies infectieuses sur le teneur en protéines sériques et les différents constituants est plus importante dès que les conditions alimentaires laissent de plus en plus à désirer.

Le facteur racial ne peut être nié, ni montré.

En ce qui concerne le régime, les recherches ont été effectuées par M^{lle} Sh. Malcolm, diéticienne nutritionniste, de la F.A.O. Les analyses sériques ont été faites par le D^{r} J. C. M. Verschure dans le Stads en Academisch Ziekenhuis à Utrecht.

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GENERAL DISCUSSION

Dr. O. Bassir (U.K.) — Mr. Chairman, I would like to ask the following questions:

1. *Platt* — Have any experiments been done to see whether the islets of Langerhans are regenerated upon the animals being put on a high-protein diet? What is the magnitude of the changes in the $\frac{\alpha}{\beta}$ cells to which the author referred?

2. Were there any special abnormalities of mineral intake in the diets of the Indonesian children showing the calcification of pancreas?

3. *Dr. Walters* — In view of the sensitivity of liver cells, both in respect to the physical as well as chemical characteristics, to alcohol fixation, what biochemical changes does Dr. Walkers mean to illustrate by his histological demonstration?

4. *Dr. Jensen* — β-globulins have been shown by Smithie's starch gel method to be genetically controlled in some humans. Has the author used such sensitive methods to see wheter genetical differences in the distribution of serum proteins are superimposed upon the infection influence which has been demonstrated upon the γ globulin?

Dr. B. Platt (U.K.) — *Answer to question 1* — We have, as yet, done no recovery experiment on animals; I cannot therefore sepeak about regeneration of the islets of Langerhans.

Answer to question 2 — In one series of animals the results for the proportion of α-cells was for controls 24,7 %, for animals on low protein diet, 41,5 %, and for those on low protein plus additional carbohydrate, 30,7 %.

Prof. A. W. Woodruff — In 1950, while investigating nutritional anaemias in Nigeria, it was noticed that the serum albumin fraction of those infected was invariably less than normal and the globulin fraction invariably increased. In 1953, when again investi-

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gating anaemias in Nigeria, the composition of this increased amount of globulin in the serum of these patients was studied electrophoretically. It was then found that the gamma fraction of the globulin was responsible for the increased amounts of globulin present and it was later shown that the serum of most Nigerians contains a greater amount of gamma globulin than does that of persons from temperate climates. This raised a number of questions regarding the origin of the gamma globulin and the three most likely causes for its presence appeared to be nutritional liver damage, hereditary factors or infection with malarial and possibly other protozoal infections endemic in the neighbourhood. With the co-operation of Dr. D. G. Davey, experiments were undertaken to determine firstly whether malarial and secondly whether trypanosomal infection could raise gamma globulins in infected animals. Care was taken to give the animals adequate diets so that nutritional and other factors could as far as possible be excluded as causes of any changes which might be found.

In brief it was found — Woodruff (1957) — that gamma globulin in the serum was increased in rats by infection with *Plasmodium berghei* and in monkeys by infection with *Plasmodium knowlesi*.

The outstanding change observed in *Trypanosoma rhodesiense* infection in monkeys has been in the gamma globulin fraction of the serum. In five monkeys in which infection continued for 20 weeks or until earlier death, the gamma globulin initially accounted for a mean of 17.7 per cent of the total serum proteins and after 20 weeks of infection for a mean of 31.6 per cent of them. In two monkeys whose blood continued to show parasites until the 30th week after initial infection the value had by then risen to 40.8 per cent. Slight reduction in the serum albumin values in the monkeys occurred, but the difference between these before and 20 weeks after infection was not statistically significant. Monkeys were given suppressive treatment with Antrypol when their condition suggested that they might not otherwise survive. In one monkey, following three such treatments, parasites permanently disappeared from the blood and the serum protein pattern remained almost constant throughout 30 weeks of observation. In spite of the absence of changes in this protein pattern of the serum, eleven attempts to reinfect the monkey with *T. rhodesiense* failed. It appears, therefore, that this animal acquired a well marked immunity to the infection although there were no significant changes detectable electrophoretically in the serum. This suggests that only a small amount of the large quantities of gamma globulin induced by infection with *T. rhodesiense* consists of antibodies for this animal evidently had sufficient antibody to prevent reinfection whereas infected monkeys continued to show trypanosomes in the blood in spite of the presence of large quantities of gamma globulin in the serum.

These experiments indicate that trypanosomal and malarial infection induce a considerable accumulation of gamma globulin in the serum and diversion of an individual's protein supplies to this gamma globulin presumably has some effect on the nutritional status. Secondly, they show that protozoal infections are capable of causing changes in the serum gamma globulin of the order of those commonly seen in tropical peoples and, in view of the widespread distribution of such protozoal infections in the tropics, it appears possible that they, rather than hereditary factors, are responsible for the increased gamma globulin values commonly found in these peoples. The possibility, however, that hereditary and nutritional factors and liver damage play some part in the production of these altered gamma globulin values cannot be excluded.

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Dr. J. H. Walters (U.K.) — Dr. Bassir has asked the very cogent question as to whether it is justified to draw conclusions as to the calibre of capillaries in life from the appearances of alcohol-fixed, stained material. I tried to make it clear that the appearance of such sections can convey only an indication of the state of the vascularity of the living organ. However, it seems justified to draw relative conclusions between two sets of material obtained, fixed and stained in an identical manner.

Dr. P. Zuidema (Netherlands) — The diet of these patients is inadequate in calories and extremely low in animal protein. In private practice diabetes is common among Indonesians from the higher levels of society and among Chinese; this is the common form of diabetes so well-known in Western countries. The «diabetes of the poor» is in marked contrast with the «diabetes of the rich».

Dr. J. R. Lauckner (Nigeria) — Dr. Zuidema, in describing the unusual type of diabetes seen in the lower class of patients in Indonesia, has almost exactly described the clinical picture of diabetes as we commonly see it in Ibadan. His communication is therefore of very great interest in suggesting that our cases of diabetes, which have puzzled us considerably, may in fact be due to protein malnutrition in earlier life.

I should like to ask him two questions:

1. How were his 45 cases selected? Did they represent all cases of diabetes seen during a certain period?
2. Has he any pathological (laparotomy or autopsy) confirmation of his presumptive diagnosis of fibrosis of pancreas, to explain the radiological calcification observed?

We have not, as far as I know, observed pancreatic calcification in our diabetics in Ibadan, but as soon as I return to Nigeria I shall certainly Xray the abdomens of as many diabetics as I can find.

Dr. H. Oomen (Netherlands) — How long will malaria pigment remain visible in liverbiopsies after the last attack of malaria?

Prof. D^{r} J. Sénécal (Dakar — A.O.F.) — 1) *À propos de l'évolution du pigment palustre dans le foie* — Les ponctions biopsiés répétées chez les enfants atteints de paludisme montrent que le pigment se présente d'abord sous forme de grains très fins disseminés dans le lobule; puis les grains se condensent formant des blocs plus volumineux et plus proches de l'espace porte. Finalement, le pigment n'est plus retrouvé que dans l'espace porte, d'où il disparaît complètement après un an ou 18 mois. La question se pose de savoir s'il s'agit d'un transfert du pigment dans l'espace porte par les cellules de Kupffer ou d'un métabolisme plus actif au centre du lobule. Un travail sur la disparition du pigment a été fait à Lyon par le Prof. Sohier et ses collaborateurs sur les sujets rapatriés d'Outre-Mer. Il a trouvé qu'après 18 mois il n'y avait plus de pigment.

2) Le *D^r^ Sénécal* demande au D^r^ Platt, à propos de lésions pancréatiques, s'il s'agit d'une augmentation réelle des cellules α ou d'une diminution des cellules β?

DR. A. J. JANSEN (Netherlands) — The analysis of the serum samples were done by paper electrophoresis in the University Laboratories at Utrech (Holland). There was no significant difference between the β-globulin levels of Biak-natives and those of Dutch residents at Biak.

PAPERS OFFERED AS FREE COMMUNICATIONS BUT NOT DISCUSSED DUE TO THE ABSENCE OF THE AUTHORS

BERI-BERI AS A PUBLIC HEALTH PROBLEM IN SOUTH-EAST ASIA

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To the public health nutritionist working in the countries where raw rice is consumed, beri-beri is still an unsolved problem, despite the fact that the cause has been known for some forty years. Time has shown that it is not, after all, a disappearing disease. Only the incidence has changed, being now very low in some areas where it was high, and rising steadily in others hitherto practically unaffected. Beri-beri is therefore of considerable health significance in some countries. Nevertheless, except for the initial intensive studies in the first two decades of this century, it has been largely neglected.

The reasons for this neglect are not difficult to find. The decline in incidence in the originally affected urban areas probably created a feeling that the disease was no longer a major public health problem (1, 2). Moreover, as will be shown, in areas where the deficiency is less acute, and medical services limited, beri-beri can cause many deaths without the existence of widespread disease being recognised. In addition, in most of the countries concerned, communicable diseases are also prevalent, and their control occupied the interests and energies of the great majority of public health officers. This is still true in many parts of the world, and reasonable progress in the prevention of nutritional diseases generally will not take place until the training of medical and health personnel concentrates less exclusively on the communicable disease hazards to public health.

This short review of the existing knowledge about beri-beri as it occurred, for example, in Malaya, and is now occurring in South-East Asia, is presented in the hope that it may disclose some of the gaps in that knowledge, and perhaps indicate how they may be filled.

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The work of Takkaki and other investigators in Japan, of Eikmman, Vorderman and Grijns in the Netherlands Indies, of Braddon and Stanton in Malaya, and of Vedder and Chamberlain in the Philippines established the true nature of the disease. In Malaya, some fifty or sixty years ago the problem was sharply defined because there were three large racial groups — Malay, Chinese and Indian — whose staple food was rice. The difference in the incidence of beri-beri in these three communities was great, and the results of the investigations into this difference increased considerably our knowledge of the public health aspects of the disease.

The indigenous Malays lived mostly around the coast, cultivated their own rice, and hand-milled it. The Chinese who came into the country in great numbers as tin miners, preferred, and imported, a very highly milled rice. The Southern Indians who provided the labour force on the rubber estates preferred a parboiled rice which also was imported.

Beri-beri at that time was a disease of the Chinese, and was responsible for enormous morbidity and mortality among the young, able-bodied labourers who were recruited in China, given a sum of money and transported to the mines in Malaya. There they were lodged in large huts and provided with a very small monthly wage, bedding, mosquito nets and food for the whole of their agreement, which might extend from six months to two years. After they had completed their agreement they were paid wages and, beyond being provided with a roof over their heads, organized their own lives. In addition to these there were the Chinese owners and foremen, etc., who were, of course, still better off financially (3).

The young apprentices got unlimited quantities of rice as part of their wages and it is stated that where very heavy labour was required, up to 2-1/2 lbs. per man per day was eaten (4).

Hamilton Wright (5) records that 800 out of 2,400 such recruits died of beri-beri in one isolated mine in a two-year period.

From the available records, it appears that the disease was not particularly common in the populations in China from which these youngsters were drawn. Braddon rightly came to the conclusion that the severe disease was due to the large consumption of highly milled rice by young men whose diet poverty had previously kept within safe limits. If later they moved to the rapidly growing towns and worked in shops, pulled rickshaws, etc., and bought their own food, they contracted the disease much less frequently. The relatively well-to-do mine owner and foreman seldom got beri-beri.

Nevertheless, among the Chinese population as a whole the disease was very common. In Kuala Lumpur — originally a mining town — there was, at the turn of the century, one general hospital with 14 wards, of which 8 were reserved for beri-beri cases (6). Braddon estimated that of every 1,000 Chinese coming into the country annually, 120 got beri-beri, 80 had it severely and 16 fatally.

Between 1900 and 1920 there was a marked reduction in the admission rate to hospital, probably due to changes in living conditions. In the earlier part of the century, there were 4-5 men to every woman and a high proportion of men were housed in labour camps. With the arrival of women from China in increasing numbers, homes were set up and vegetable gardens and other local food production became established. Roads were built which reached most of the remote mines, the transport of food became much easier and the degree of imbalance in respect of thiamine was greatly decreased.

Conditions became stabilized except for two pre-war periods when an increased incidence occurred — both during times of greater prosperity. The annual reports of the Director of Medical Services constantly refer to the effect of improved economic circumstances in increasing the incidence of beri-bri, and incidentally the infantile mortality rate.

In times of prosperity the disease was particularly prevalent among smallholders. When they could sell their rubber for a good price, they also sold their rice and bought the greatly preferred highly milled, best Siam rice. At other times, when the smallholder had to economize, he hand-pounded and consumed his own rice. Prosperity therefore introduced the disease into communities where previously it did not exist, or was at least relatively rare. A similar beri-beri potential exists today in the rice growing communities in certain other countries and is being activated by the introduction of small power-driven rice mills.

With increasing prosperity and sophistication, these small mills are rapidly spreading into the rural areas and the practice of hand-milling is giving way to machine milling. Only limited surveys have so far been carried out in these areas, but they indicate that in some at least, there is quite a high incidence of disease.

In one area in North Thailand, for example, some 24 per cent were found to have neuropathies attributable to beri-beri (7). Reports from other areas in this region mention outbreaks of beri-beri three months after power mills had been introduced.

The chief importance of this trend lies in its possible effect on the infant and this aspect of the total problem urgently needs clarification. For several reasons there has never been an adequate assessment of the importance of beri-beri as a cause of mortality in the infant.

Towards the end of the last century Hirota (8) described a clinical entity in the infant which he recognized as beri-beri. After this infantile beri-beri received quite a lot of attention in Japan, though it does not appear to have been of major significance as a cause of death (9).

In the Philippines, however, the infantile form of the disease was thought to be the main cause of the very high infantile mortality. In 1904 Guerrero (10), on the basis of clinical observations, concluded that the disease known as «taon», which was responsible for such a large number of infant deaths, was, in fact, beri-beri.

Subsequent investigations carried out by McLaughlin & Andrews in 1910 (11) verified this conclusion. In autopsies on infants under one year they found that 56 per cent had died of beri-beri, although, in many cases, that diagnosis had not been made prior to death. The infantile mortality in Manila in that year was 488 per mille. They concluded that the unusally high mortality rate in the early months of life in the *breast-fed infant* was almost entirely due to this disease. Andrews in 1912 (12) laid stress on the age at death as an important characteristic of the disease. He found that nine-tenths of the deaths occurred between the ages of one and three months; that the onset of the disease was often unnoticed by the mother because of the mild early signs; and that when frank condition developed the course was rapid and frequently ended fatally. The occurrence of sudden death was also noted. He showed that the maternal milk was responsible for the death of the infants, for he was able to produce beri-beri in puppies by feeding them on the milk of women whose infants had died of beri-beri.

About this time Vedder & Chamberlain (13) developed an extract of rice polishings known as «tikitiki» extract, which proved effective in the treatment of the mild case and was later used in prevention.

The problem of infantile beri-beri in the Philippines was followed closely from 1908-47 by Albert (14, 15). In 1931 he reported that the introduction of the extensive use of tikitiki had reduced the infantile beri-beri mortality from 93.66 per 1,000 births in 1915 to 19.12 per 1,000 births in 1930 (16). In 1933, 1934 and 1935 there was an increased incidence (17). In the latter part of the war period, and immediately after the war, no cases were admitted to hospital. However, in 1947 cases again appeared. In his survey of beri-beri in the Bataan, Salcedo (18) found few cases of infantile beri-beri in his clinical survey of the population, in spite of the fact that the majority of the total deaths from the disease occurred in infants.

Reports from a number of other countries indicate that infantile beri-beri has been an important cause of morbidity and mortality.

Bray (19) described the disease in a small population under medical supervision in Nauru Island in the Pacific.

In Malaya, frequent reference is made in pre-war reports to the contribution made to the high infantile mortality by this disease. Haridas (20) *et al.* (21, 22) records the admissions of over 300 cases — nearly 11 per cent of all admissions to the children's wards, in the General Hospital, Singapore.

Aykroyd & Krishnan (23) drew attention to the high infantile mortality rates in certain areas in what was then called Madras Presidency — with a high proportion of children dying between two and five months. They considered that this evidence was suggestive, but not proof, of a high death rate from beri-beri.

Krishnan, Ramachandran & Sadhu (24) treated over 1,000 cases of infantile beri-beri in the same area, but did not attempt to find out the proportion of infants that developed beri-beri, or the quantitative effect of the disease on the infantile mortality rate.

In Hong Kong and the Netherlands Indies before the war, and more recently in Indo-China (25), the existence of a health problem produced by infantile beri-beri was well recognized. Féhily (26) states that 18 per cent of the infants attending infant welfare centres in Hong Kong showed signs of infantile beri-beri, and that the mortality rate in untreated cases was over 90 per cent.

Sharma (27) describes the many cases of infantile beri-beri which he saw during his 40 years as a paediatrician in Burma, and the practices and dietary habits which were their unvarying accompaniment. Reports from a WHO consultant support the view that beri-beri is still an important cause of infant deaths in that country.

Some of the publications suggest that a very high infant death rate results, and it may be that this can occur under some circumstances and not under others. This is clearly a matter on which exact information is required. For several reasons, the total problem of the public health significance of infantile beri-beri is at present difficult to define. In the pre-war period the registration of births and deaths was far from complete in most of the countries affected, and there was therefore no reliable information as to the total number of infants dying. Also, at that time, the attention of health authorities was concentrated en communicable disease which was an ever-present menace to the health of the total population. In addition to this, the people among whom the disease most often occurred were generally unsophisticated, little exposed to modern developments and naturally inclined to consult their own traditional healers rather than the trained practitioner. Lastly, there appears little doubt that the disease was often not diagnosed.

At the present time some of these circumstances remain, especially in the rural areas.

Moreover, it is essentially the rural populations that are now being exposed to the risk of an increased incidence of both adult and infantile beri-beri, due to the spread of small rice-milling machines throughout the countryside. This change-over from an undermilled to a highly milled rice is taking place in populations which derive a very high proportion of their calories from rice. The medical services to these communities are limited and may not be easily accepted, and transport is usually difficult. Rural people in rice-growing countries usually live in small villages approachable only over paths on the tops of the bunds in the rice fields which in the wet weather may be flooded and are always difficult to negotiate. This last point is one of considerable importance, because beri-beri is a seasonal disease in many countries — with a higher incidence during the rainy season. Under these circumstances there is likely to be difficulty and delay in getting children to the hospital or clinic.

Infantile beri-beri is often an acute disease. Andrews (28), Bray (29), Féhily (30) and Sato (31), among others, have stressed its general acute nature and the frequent occurrence of sudden deaths — a feature which, in the breast-fed infant, the last writer regards as being pathognomonic of infantile beri-beri.

Haridas (32), in a review of 108 cases, found signs of cardiac disease in 94 of them and even with treatment, a high death rate. In circumstances of comparable severity in remote villages with poor communications, it is unlikely that any of the affected children would be seen and the disease diagnosed. It is therefore perhaps not difficult to understand why a reasonably accurate assessment of the situation has not yet been achieved.

It is not nearly so difficult to assess the prevalence of the disease in the adult, for it is, in the main, a chronic condition and the incapacitated individual may ultimately find his way to the clinic or hospital. In addition, the milder signs of the disease can be picked up in a clinical survey of the affected population. Obviously, if the incidence of the disease in the adult could be related to its incidence in the infant, an assessment of he public health significance of beri-beri would be a good deal easier.

Certain factors, however, may influence the accuracy of the picture of this relationship. Restriction of the maternal diet for 44 days after the birth of the infant is a very common practice in most of the countries concerned. The woman, already probably on an inadequate intake of thiamine, undergoes the stress of delivery and then subsists for six weeks on a diet of rice and dried fish, sometimes, as Sharma describes, with a high intake of sugar. This practice must, and does, in fact, expose her infant to a very considerable danger. The lactating woman and her infant are therefore at greater risk than other members of the community, although the recorded incidence in the total population may not be high (33).

General habits of living also may have their influence. The improved status of women in Japan is one of the reasons given for the decrease in infantile beri-beri in that country. The women now feed with the men and children and have access to all the foods available to the family instead of, as formerly, only the left-overs (34).

Another factor is the inconstant relationship between the disease in the mother and in her infant. It might be possible to carry out extensive investigations in lactating women in order to forecast the probable occurrence of beri-beri in the child. Hirota, however, pointed out that in some of the first cases of infantile beri-beri he described, no signs of the disease were to be found in the mothers, until some weeks after their infants had died. Bray found little evidence of maternal beri-beri, although the disease in the infant was acute and usually fatal. Haridas found signs in only 25 per cent of the mothers of infants admitted to his wards. Féhily drew attention to the absence of any sign of maternal disease in many acute cases of infantile beri-beri, and suggested that during the pre-clinical stage the mother would probably lactate well and the child would therefore be more likely to acquire the disease. The development of the frank disease would interfere with lactation and the risk to the child would then be less. Beri-beri in the mother, without the child showing any sign of it, has also been recorded.

For these reasons it is difficult to form any opinion as to the relationship of the occurrence of the disease in the mother and in her child and further investigations into the whole problem are necessary.

One possible approach might be to carry out pilot studies in two similar areas — one where the rice is highy milled and another where it is handpounded — to establish the differences in infantile mortality rates. In addition to the total births and infant deaths in these two areas, the exact age at death and the mode of death would have to be ascertained, the sick infants seen by a paediatrician, dietary surveys to establish the degree of imbalance, and clinical surveys of groups of adults carried out.

After an investigation of this sort, it might be possible to establish a relationship between the deficiency of thiamine in the diet and the disease in the adult — possibly with special reference to the lactating woman — and the infantile mortality associated with these more readily measurable entities.

Another approach to the problem might depend on the assessment of the thiamine nutriture of the lactating woman. Some attention is now being paid to the level of thiamine in breast milk.

A great deal has been known about beri-beri for many years, but if the full significance of beri-beri as a public health problem is to become clear, and future tragedy avoided in South-East Asia, the gaps in our knowledge must be filled.

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CHARACTERISTICS OF PELLAGRA IN SERBIA

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As pellagra has its specific characteristics in every country where it occurs, and as it has an individual character as well, we are of the opinion that it would be useful to acquaint ourselves with the specific aspects of pellagra in Serbia, as a contribution to the solution of the pellagra problem.

Pellagra has been existing for many decades in the Kosovo-Metohija region in Western Serbia. This region is inhabited by Serbs and Shiptars. Agriculture is almost the exclusive occupation of the inhabitants, and the people are mostly poor. Their diet is unbelievingly stereotyped. Pellagra is distinctly a chronic disease in this region. It is not rare to find persons who have been suffering from pellagra from their youth to old age. The incidence of this disease is greater among Shiptars (mostly Moslems) than among Serbs (exclusively of the Orthodox faith). Adults contract the disease between the ages of 40 and 50 and children between 10 and 15. Boys contract it more often than girls. The number of observed cases of pellagra exceeds 2,000.

Considering that this disease is widely spread, it is of special interest to examine the diet of the people in this region.

Results of the investigations carried out by K. Snajder, of our Institute, show that of the total caloric value, 75.6 % of the diet of the Serbs is covered by *bread*, and of the Shiptars 76.8 %. The annual average of *bread, maize bread* among Serbs covers 75 % and among the Shiptars 93.5 %. *In the total caloric value of the diet*, maize

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bread is represented, among Serbs, by 57.2 % and among the Shiptars by 71.9 %, while wheat bread among Serbs is represented by 23.2 % and among the Shiptars by 6.5 %. The annual average of victuals consumed per one member in one day consists of 50.8 g of proteins, of which 5.8 g are animal, 35.6 g of fats and 336.7 g of carbohydrates.

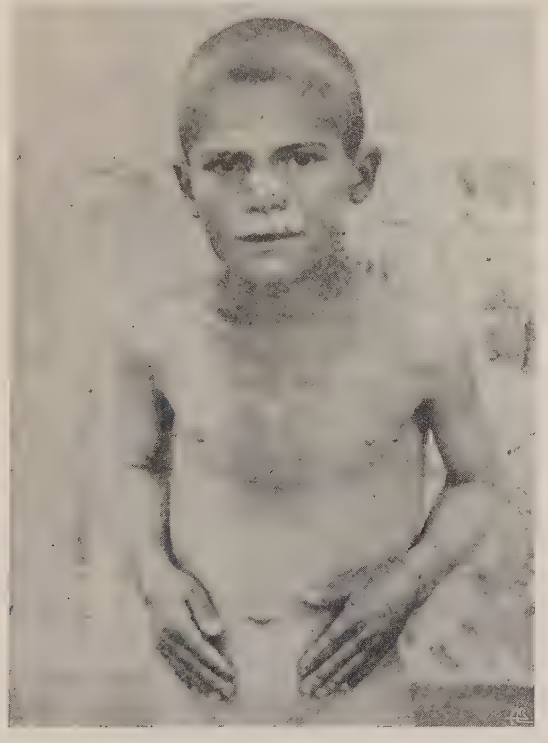

Fig. 1 — A. S., aged 10, Prekas. Pellagrous changes on the nose, pellagrous «gloves». Casal's necklace

As can be seen, the diet of the population in this region is preponderantly carbohydrate in character, with insufficient percentages of proteins, especially animal, and insufficient fats. As to the consumption of vegetables, the diet is deficient because it has a seasonal character, and is absolutely standardized.

The large percentage of maize flour in the diet of the population of this region with insufficient quantities of proteins of animal origins undeniably points to the role of maize as an etiological factor in pellagra.

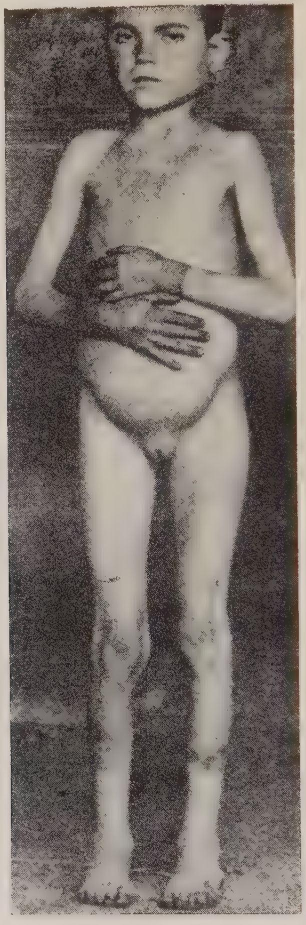

Fig. 2 — R. R., aged 8. Duration of illness 5 years. Multilocularity of hyperpigmented pellagrous changes. Apathetic expression

From our experience we know that, even at the absolute maize diet, pellagra will not appear if the minimum, at least, of animal proteins is provided in that diet.

In Serbia pellagra appears in principle in the form of a distinctly chronic disease which with its rhythmical aspect very often lasts throughout life, beginning in youth. These persons, however, are only medically, but not socially ill (Fig. 1). Contrary to this type of pellagra there are patients with classical symptoms whose state is much graver. They are the cases with dull, somewhat apathetic expression, inhibited movements and slow reactions, pale yellow skin, and they give a general impression of heavily intoxicated persons. To designate this condition as a demented state is, in our opinion, incorrect (Figs. 2 and 3).

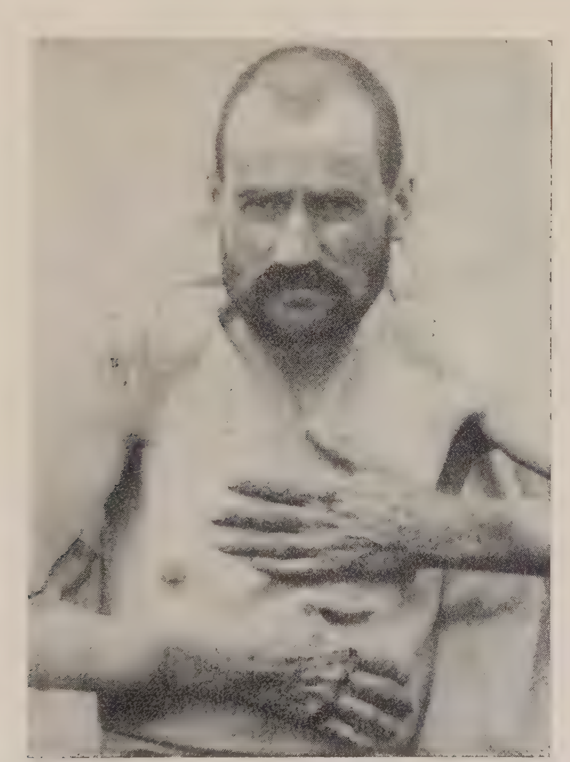

Fig. 3 — A. I., aged 44, Globare. Duration of illness 3 years. Rather long pellagrous «gloves». Changes on the skin of the chest and face. Haevy adynamia

The explosion of the disease under the influence of the first spring sunshine is not preceded by any long period of uneasiness. «Temperature», which is nothing else but inflammation of the skin on the body, poor sleep, nervousness, often diarrhoea appear.

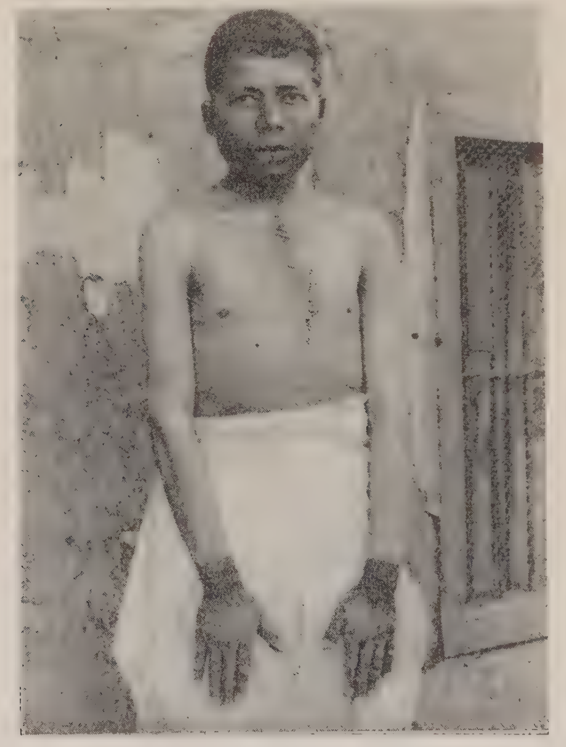

Fig. 4 — R. S., aged 27, Bela Crkva. Short, black pellagrous «gloves»

The clinical picture of pellagra in Serbia is not basically different from the one classically described. It is interesting to note that the pigmentation of unprotected parts of the body sometimes takes such proportions that the respective person assumes the distinctions of another race (Figs. 4 and 5).

Psychoses are rare: only 7 cases were found in 2,085 patients with pellagra. Diarrhoea was absent in 30 % of cases, nor was there a single case of macrocytic anaemia.

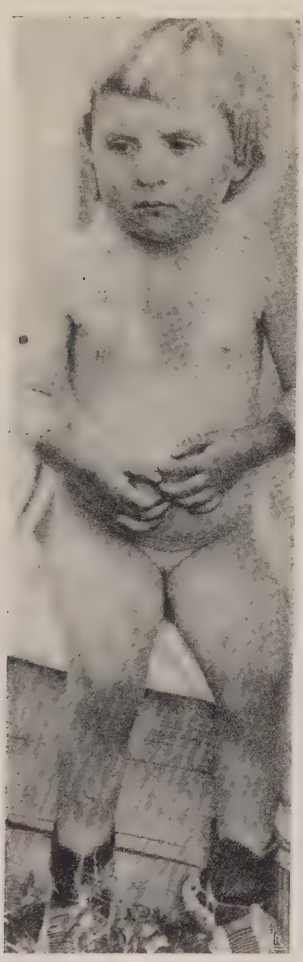

Fig. 5 — N. N. Long, black pellagrous «gloves»

As for changes on the lips and tongue, that component part of the clinical picture of pellagra, investigations of several thousand rural inhabitants and industrial workers in Serbia have shown that identical changes, although of lesser intensity, can also be found where pellagra is not involved at all.

Another symptom, which we have not found in the available literature, concerns the changes on the soft palate and uvula in the form of bullose efflorescences. Likewise, we did not come upon the injection of individual or groups of blood vessels of the soft palate, similar to the vascularization of the conjunctiva in ariboflavinosis

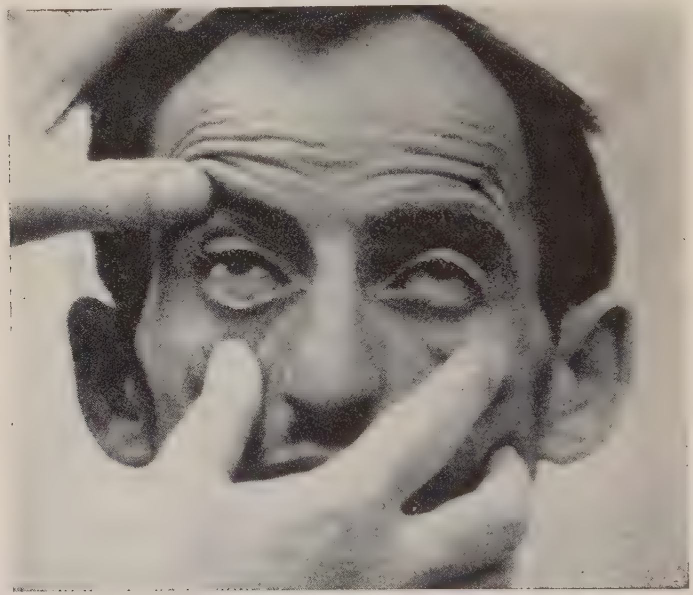

Fig. 6 — J. B., aged 28, Rakos. Vascularization of conjunctivae bulbi. Pericorneal injection. Muddy cornea. Changes on the cornea Photophobia. Symptom of the «scarf»

(Fig. 6). We have arrived at the conclusion that the deficiency of animal albumen of meat and milk is the cause of all these changes. There is a regular relationship between the length of non-intake of these foods and the degree of intensity of the symptoms. Changes on the soft palate are undoubtedly one of the first symptoms of this lack. It is important to note that these changes do not cause any discomfort to the patient.

Injection of the blood vessels of the conjunctiva and pericorneal injection fairly frequently offers the picture of a *ribbon or scarf*, as we have termed this state. It is a question of the vascularisation of the aperture of the apple of the eye with photophobia, a symptom frequently found in children's pellagra. It is possible to notice from quite a distance the interesting and impressive expression of the eyes of these patients.

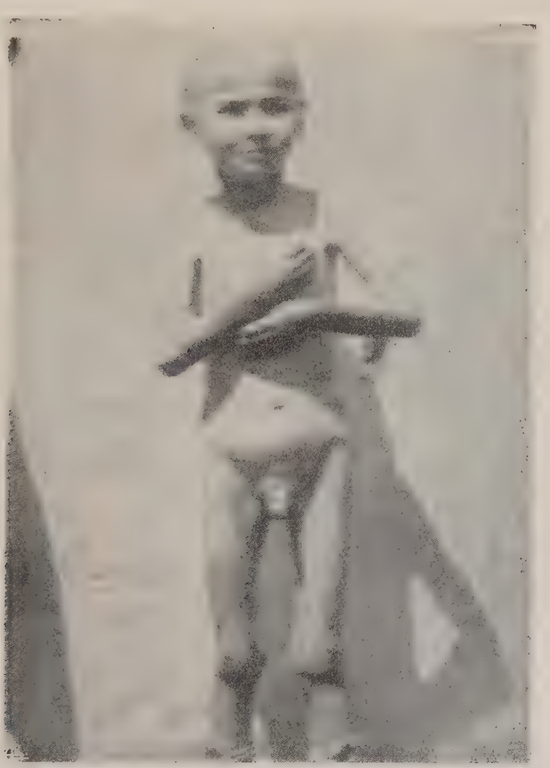

Fig. 7 — H. H., aged 8, Ukča. Ilness of three years duration. Infantile pellagra of the same type as that of the adults. Catastrophic retardation in growth

Children's pellagra has the form of the adult pellagra and is followed by retardation in growth, often of catastrophic proportions, of the diseased children (Figs. 7 and 8).

Pellagra has serious consequences for women and is accompanied by disturbances in the menstrual cycle, gravidity and lactation.

Although primarily a social problem, pellagra is undoubtedly also a medically interesting one. It is the consequence of the nature of many of the deficiencies and, in our opinion, the form that the disease will take clinically depends on the degree of these deficiencies.

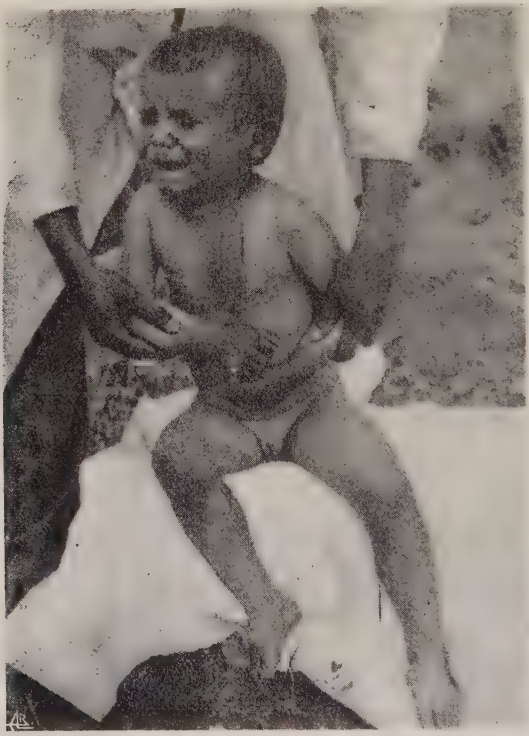

Fig. 8 — K. S., aged 4, Crkolez. Illness of two years duration. Infantile pellagra of the same type as the pellagra of the adults

Differences in the clinical picture of pellagra in various countries should be regarded in the light of national diets in each country.

The biological feature of the kernel of maize, with little tryptophane as the source substance for nicotinic acid, is able to compensate for at least the minimum of albumen of animal origin in the diet, as is confirmed by many of our investigations in various parts of Serbia, where maize is the chief food.

Just as maize is of great significance in the problem of pellagra, the presence of animal albumens is an equally important factor in understanding the problems of this disease.

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INFLUENCE DE L'ALIMENTATION SUR LA DYSPROTÉINÉMIE DE L'AFRICAIN

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En étudiant les résultats des électrophorèses sur papier de sérum sanguin d'Africain, nous avons été frappés par l'importance de la dysprotéinémie rencontrée:

Sur 2.375 sérums examinés, dont 504 sérums d'enfants, on trouve une protéinémie moyenne de 74,7 avec des taux d'albumine de 45 % et de 27 % de γ globuline. Ces sérums ont été prélevés chez des sujets sains au cours d'enquêtes systématiques en brousse.

Cette hypergamaglobulinémie et cette hypoalbuminémie se rencontrent dès l'âge de 3 ans aux mêmes taux que chez l'adulte. Nous n'avons pas étudié la protidémie des nouveaux-nés et des nourissons, mais les études de Close d'une part et de Thompson d'autre part ont montré que les taux d'albumine et de globulines sériques sont analogues à ceux des nouveaux-nés et des nourrissons européens. L'hypergammaglobulinémie apparaît entre 1 et 2 ans.

De nombreuses hypothèses ont été avancées pour expliquer cette dysprotéinémie. Pour certains il s'agirait d'un facteur racial, d'autres y voient le reflet d'une alimentation végétarienne, d'autres, enfin, en font la traduction de l'infestation palustre.

Pour notre part, nous avons recherché au cours d'enquêtes systématiques les relations existant entre l'alimentation et cette dysprotéinémie commune de l'Africain. Nous avons par ailleurs étudié les

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modifications éventuelles apportées à cette dysprotéinémie par une supplémentation protidique.

Les enquêtes alimentaires jointes à l'étude des sérums sanguins ont été effectuées au Moyen-Congo dans trois districts: un district de forêts: Mossendjo, un district de savane: Mindouli, et un district situé au bord de la mer: Madingo-Kayes.

Les enquêtes alimentaires ont été effectuées par pesée des aliments avant la cuisson. Les poids obtenus étaient reportés sur des carnets d'enquête où figuraient les noms des rationnaires de chaque repas. On a ainsi étudié l'alimentation de 186 familles suivies chacune pendant une semaine, ce qui représente 1.300 journées d'enquêtes et plus de 2.500 repas pesés.

Nous ne donnerons ici que le taux per capita de protéines totales et de protéines animales. Ces taux ont été calculés en utilisant les tables de composition des aliments éditées par la F.A.O. en 1954, sauf pour le manioc, le poisson salé et la viande boucanée, pour lesquels nous avons utilisé les résultats de nos propres analyses.

L'étude des protéines sériques a été faite par réfractométrie contrôlée par le dosage de l'azote total et par électrophorèse sur papier.

On a ainsi obtenu les résultats suivants:

	Nombre de familles étudiées	Ration jour per capita		Nombre de sérums étudiés	Protéines sériques (g/l)	Gamma-globulines (%)
		Protéines totales (g)	Protéines animales (g)			
Mindouli	58	40	10	115	75	33
Mossendjo	96	48	14	82	77	36
Madingo-Kayes	32	45	28	203	78	37

Le taux de protéines sériques et de gammaglobulines est sensiblement le même dans les trois districts, alors que l'apport protidique varie et que surtout les protéines animales sont trois fois plus élevées à Madingou-Kayes qu'à Mindouli. Ces résultats montrent qu'il n'y a aucune relation entre le taux des gammaglobulines et la quantité ou la qualité des protéines alimentaires.

Afin de confirmer ces résultats, nous avons effectué des examens de sérums chez des écoliers de la région de Brazzaville avant et après supplémentation protidique.

Nous avons utilisé deux sources de protéines. Dans une école, la supplémentation a été effectuée avec du lait écrémé sucré en poudre, dans les autres avec des arachides crues ou grillées. Ainsi, il nous était possible d'apprécier, d'une part, l'action des protéines animales et, d'autre part, celle des protéines végétales.

Dans l'école recevant du lait l'expérience a porté sur 48 enfants âgés de 5 à 12 ans qui ont reçu chaque jour pendant trois mois 50 g de lait écrémé sucré en poudre. Ce lait leur apportait par jour:

Calories	Protéines (g)	Lipides (g)
194	10	0,5

Le chifre de 48 enfants correspond à ceux qui ont consommé du lait pendant les trois mois.

En effet, au départ, le chiffre des enfants supplémentés était de 102, mais rapidement nous avons dû limiter la distribution de lait aux élèves volontaires en raison de phénomènes diarrhéiques accompagnés d'amaigrissement parfois important. Chez les 48 enfants restants l'effet sur l'état général et le poids a été excellent.

Par contre, le taux de protéines sériques est resté inchangé, ainsi que le taux d'albumine et de gammaglobuline, comme le montre le tableau suivant:

Enfants ayant reçu du lait écrémé pendant trois mois

	Nombre de sérums étudiés	Protéines sériques (g / l)	Albumine (%)	Gamma-globulines (%)
Avant supplémentation	65	73,5	47	29
Après supplémentation	48	73	48	28,2

Dans deux autres écoles de la même région la supplémentation a été réalisée par des arachides crues ou grillées.

Pendant trois mois on a distribué chaque jour à chaque enfant 50 g d'arachides décortiquées qui apportaient:

Calories	Protéines (g)	Lipides (g)
273	12,8	21,6

Cette supplémentation portant sur 215 enfants de 5 à 12 ans a été très bien acceptée par tous, et, malgré l'important apport lipidique, elle a été remarquablement bien tolérée. Les résultats sur l'état général et le poids ont été analogues à ceux obtenus avec le lait écrémé. Il en est de même pour les protéines sériques et les gammaglobulines, qui, là également, sont restées inchangées, comme le montre le tableau suivant:

Enfants ayant reçu des arachides pendant trois mois

	Nombre de sérums étudiés	Protéines sériques (g/l)	Albumine (%)	Gamma-globulines (%)
Avant supplémentation	47	76	49	29
Après supplémentation	48	74	47	29

Ainsi, qu'il s'agisse de protéines animales ou végétales, une supplémentation de 10 g par jour pendant trois mois n'a amené aucune modification du taux d'albumine ou de gammaglobuline sériques chez les enfants étudiés.

Ces résultats confirment ceux que nous avions obtenus avec les enquêtes alimentaires et tendent à prouver que ni la quantité, ni la qualité des protéines n'influent sur la dysprotéinémie de l'Africain.

Au cours de nos enquêtes le seul facteur qui nous a semblé varier parallèlement à cette dysprotéinémie était le taux de splénomégalies traduisant l'intensité de l'infestation palustre, comme nous le voyons sur le tableau suivant:

	Splénomégalie		Protidémie		
	Nombre d'enfants examinés	(%)	Nombre de sérums examinés	Protides totaux (g/l)	Gamma-globulines (%)
Brazzaville	2 577	12	1.877	74,3	26
Kibouende	207	22	204	75	29
Mossendjo	4.202	35	82	77	36
Madingou-Kayes	1.066	40	203	78	37

Ainsi, à Brazzaville, où le taux de splénomégalie est faibls en raison de la lutte imagocide, le taux de gammaglobuline est de 26 %, alors qu'il passe à 37 % à Madingo-Kayes, où l'endémicité palustre est importante.

En conclusion, les enquêtes alimentaires jointes à des essais de supplémentation protidique nous ont montré que ni la quantité, ni la qualité des protéines alimentaires ne semblent influer sur la dysprotéinémie de l'Africain.

Le paludisme est peut-être responsable de l'hypergammaglobulinémie, car c'est le seul facteur qui varie paralèlement à cette dysprotéinémie.

RÉSUMÉ

À la suite d'enquêtes alimentaires portant sur 186 familles et d'analyses de 400 sérums sanguins, les auteurs mettent en parallèle le taux de protéines alimentaires et celui des protéines et des gammaglobulines sériques. Ils montrent qu'il n'existe aucune corrélation entre ces taux.

Ils ont, en outre, effectué des examens de sérum sanguin chez des enfants avant et après supplémentation protidique. D'une part, 48 enfants ont reçu chaque jour pendant trois mois 10 g de protéines animales d'origine lactée, d'autre part, 48 enfants ont reçu chaque jour pendant trois mois 12 g de protéines végétales tirées de l'arachide. Les taux de protéines sériques, d'albumine et de gammaglobulines sont restés inchangés.

Par ailleurs, les auteurs ont constaté un certain parallélisme entre le taux de gammaglobuline et le degré d'infestation palustre.

Ils concluent que ni la quantité, ni la qualité des protéines alimentaires n'influent sur la dysprotéinémie de l'Africain, qui paraît par contre lié au paludisme.

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ASPECTOS CLÍNICOS DE ALGUNS CASOS DE *KWASHIORKOR* NA CIRCUNSCRIÇÃO DE MUTARARA, ÁFRICA ORIENTAL PORTUGUESA

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INTRODUÇÃO

A circunstância de termos podido assistir a duas épocas de fome calamitosa na área da Delegacia de Saúde, sobretudo no Posto Administrativo de Inhangoma, motivadas por grandes cheias do rio Zambeze e seus afluentes, que destruíram mais de 90 % das culturas indígenas, indo nalguns pontos aos 100 %, proporcionou-nos uma oportunidade de observar alguns casos de *kwashiorkor*, quer nas suas formas de uma rica sintomatologia, quer nas suas formas mais frustes, de sintomatologia mais discreta.

Ali vive uma população nativa de 60 000 almas, com uma densidade demográfica razoável.

A terra é plana, fértil e, nalguns pontos do interior, mesmo abaixo do nível do mar.

A ilha de Inhangoma tem uma superfície de 10 km².

Só numa parte do ano, durante as cheias, é que é uma ilha no sentido geográfico do termo; na época das secas está ligada ao continente. É que o rio Zambeze, na época das cheias, espraia-se por um braço que fecha um ângulo fluvial, cujos dois lados são constituídos pelo próprio rio Zambeze e pelo rio Chire — rios de curso permanente —, braço que forma temporàriamente um novo rio, de curso temporário: o Ziu-ziu.

A terra, sempre que não há uma estiagem prolongada, ou então quando há demasiadas chuvas, é ubérrima.

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Por este motivo, os principais produtos alimentares podem dar por vezes três produções anuais; e assim o nativo é apegado à terra que lhe dá de comer.

Porém, se há anos agrícolas óptimos, pela pujança do seu solo, noutros, ingratos e desoladores, o indígena sofre de fome.

Apesar de tudo, pela sua resignação fatalista, o indígena aí se mantém, esquecendo-se mais dos anos maus que dos bons; e daí o conservar-se sempre na sua terra, que nem sempre lhe é propícia.

Pelas consecutivas concentrações anuais que fomos fazendo, para avaliarmos do estado sanitário geral da população, verificámos nuns anos o seu bom aspecto de nutrição e noutros, pelo condicionalismo telúrico, o contrário.

O nativo desta região dedica-se, na sua generalidade, à agricultura; bem escassa minoria dedica-se ao pastoreio de animais domésticos.

Vive em agrupamentos tribais primitivos, segundo os seus usos e costumes tradicionais.

O sistema de agricultura, ainda refractário aos ensinamentos que os Europeus pretendem impor, é, por isso, simples e primitivo, e nesse caso sujeito às contingências.

Cultivam o milho, a mapira, a meixoeira, o feijão cafreal e a batata doce. O predomínio vai para o milho, que, por isso, constitui a base da sua alimentação. Por processos rudimentares de trituração e escolha, transformam-no em farinha. Este produto é tradicional na sua alimentação. Juntamente com esta farinha de milho, de que fazem uma papa, constituindo o que chamam o *caril*, comem feijão, folhas verdes, sal e, de quando em quando, carne ou peixe seco, porque são refractários à caça e à pesca.

O tipo da sua alimentação é, como sempre acontece com os indígenas africanos menos evoluídos, predominantemente vegetariano, com base na farinha de milho ou de mapira, consoante as regiões.

Acessòriamente, o indígena come as frutas da época, sem grande preocupação em as introduzir na sua alimentação diária. Daqui resulta que, quando lhes falta produto-base, por virtude de as suas machambas, que são quase sempre litorâneas, serem destruídas pelas cheias, ràpidamente se verifica um estado de desnutrição acentuado, o qual, progredindo, se traduz tantas vezes em edema de fome.

A acrescentar, aparecerem avitaminoses consecutivas também.

Nos lactentes e infantes o quadro clínico da sua deficiência alimentar prolongada traduz-se pelo aparecimento de uma sintomatologia profeiforme de *kwashiorkor*, sempre mais rápido que no adulto, como é fácil de compreender.

Durante o período de aleitamento as mães indígenas, por virtude da fome que passam, atingem estados de subnutrição bem marcados e, na sua maioria, ficam esqueléticas. Como não têm possibilidades, na grande maioria dos casos, de adquirir leite, e ainda por desconhecerem o uso dele a partir dos animais domésticos que possuem (cabra ou vaca), começam, como recurso, a alimentar os seus filhos com a alimentação própria do adulto, quer na qualidade quer na confecção.

Daí um desequilíbrio na parte digestiva do lactente, que ainda não está bem adaptado para aquele tipo alimentar do adulto, dando origem, como muito bem se compreende, a transtornos nutritivos, agudos ou crónicos.

É o seguinte o regime alimentar das crianças indígenas nos primeiros dois anos de vida:

A sua alimentação é constituída pelo leite materno; por volta dos 3 a 4 meses de idade começam já com uma papa, quer de milho quer de mapira, rala, que engolem quase sempre à força; pelos 5 ou 6 meses começam a receber a alimentação do tipo do adulto, sem passar por qualquer transição. Vão mamando sempre, enquanto a mãe tem leite, muitas vezes até os 2 ou 3 anos de idade. Chegámos a ver alguns casos de aleitamento tardio em crianças de 4 e 5 anos, crianças estas que, já comendo de tudo, mamavam o escassíssimo leite materno.

Quer dizer: sempre que a mãe tem falta de leite e, na maioria dos casos, por infra-alimentação, a criança entra ràpidamente, e sem qualquer cuidado, na alimentação dos pais.

Como dissemos já, a população nativa desta região está, no que diz respeito aos seus meios de subsistência que retira do solo, sujeita às condições climáticas, que influenciam decisivamente as suas culturas alimentares.

O condicionalismo periódico e anual das cheias, que tantas vezes atingem aspectos verdadeiramente catastróficos, transbordando demasiadamente e inundando aquelas regiões, dá por esse motivo origem a anos agrícolas nulos do ponto de vista alimentar.

Por consequência, fácil será de ver nestes dois factores, sempre ligados e na origem um do outro, a consequência inevitável e inexorável da decadência orgânica de origem alimentar que aparece nas crianças nativas.

Estes factores naturais que referimos atrás, decorrentes dos anos maus nas colheitas alimentares, consequência imediata quer de factores geográficos quer da erosão, «um factor terrível da vida alimentar africana», dando como consequência uma escassez alimentar nos indígenas desta região, depauperando a mulher durante a gravidez e durante o período de aleitamento, aliados ainda a uma alimentação exclusivamente láctea, a que estão submetidas as crianças nativas durante muito tempo, são as condições essenciais do aparecimento deste síndroma de malnutrição proteica nas referidas crianças.

Verificámos ainda, no decurso das nossas observações, que na realidade este síndroma tem uma maior incidência nos 6 meses de idade e nas alturas em que se deve iniciar progressivamente o desmame e juntar-se uma dieta adicional e, com o tempo, passar ao regime alimentar do adulto.

ASPECTOS DO QUADRO CLÍNICO GERAL

No cap. I, que trata dos aspectos médicos do *kwashiorkor*, de *El Kwashiorkor en África*, publicado em 1951 pela F.A.O., apresentam-se 8 características, que passaremos também a enunciar:

1 — Atraso do crescimento durante o último período de lactância, desmame e período seguinte.
2 — Alteração da pigmentação da pele e do cabelo.
3 — Edema.
4 — Infiltração gordurosa, necrose celular e fibrose do fígado.
5 — Alta mortalidade, independentemente do tratamento dietético apropriado.
6 — Frequente associação com várias dermatoses.
7 — Perturbações gastrintestinais.
8 — Irritabilidade e apatia mental.

Foi baseados nestas características fundamentais que fizemos o diagnóstico dos casos que vamos apresentar.

ALGUMAS CONSIDERAÇÕES

ATRASO DE CRESCIMENTO.

Verificámos, pelo gráfico das curvas de crescimento que conseguimos apurar nas crianças que frequentavam o nosso dispensário, que há, em relação às curvas de crescimento de crianças europeias, um atraso sobre este aspecto, sobretudo a partir da idade dos 8 meses.

Verificámos mais que o mesmo se passa quanto ao peso. Estas duas circunstâncias são consideradas fundamentais por Trowel, que diz: «nunca vi nenhum caso de *kwashiorkor* no qual o crescimento não estivesse consideràvelmente retardado.»

Assinalaremos, no entanto, que, apesar destes desvios no peso e na estatura das crianças com esta doença, o seu aspecto geral não é de enfraquecimento, nem tão-pouco de inanição.

Estas duas condições influíram decisivamente para que não pudéssemos internar ou conservar sob vigilância certas crianças indígenas, cujas mães recusavam o internamento dos seus filhos, quando ocasionalmente os traziam às consultas, pelo motivo de uma diarreia, uma conjuntivite, uma doença de pele, etc. Preocupavamo-nos com o aparecimento tardio de um *kwashiorkor,* pois que os casos, embora em fase ainda pré-edematosa, já denunciavam, por outros sintomas menores, o aparecimento futuro da doença. E, assim, na impossibilidade, por recusa, do internamento, as crianças regressavam ao seu meio e as mães, na sua ignorância e descuido, eram causadoras involuntárias da progressão da doença, que, sem tratamento, iria inexoràvelmente caminhando para um estado irreversível e sem remédio possível.

Mais tarde, por inquérito que efectuámos, verificámos pelo depoimento das mães que os seus filhos tinham morrido, quando acaso traziam um novo filho à consulta, pois nunca compreendiam que o tratamento estava menos nos medicamentos do que num regime alimentar correcto e próprio da idade dos seus filhos.

DESPIGMENTAÇÃO.

Este sintoma é designado para indicar uma diminuição na alteração de cor dos cabelos, ligada a deficiências nutritivas, bem como a alteração da pigmentação da pele.

No que diz respeito aos cabelos, este sintoma foi sempre constante nos casos observados e os indígenas destas regiões relacionam-no com o aparecimento da época da fome. Não verificámos, no decurso dos nossos contactos com a população indígena, quer adulta quer infantil, que pudesse ser devido a outras causas, designadamente com factores climáticos.

Verificámos, sem dúvida, que, além da despigmentação, muitas vezes os cabelos se apresentavam desfrisados e se tornavam ralos, de tal modo de nalguns pontos pareciam manchas de alopecia.

A despigmentação da pele, que verificámos nalguns casos, era sempre devida a hiperpigmentação, que não só fazia parte de algumas dermatoses, que em várias crianças abarcavam uma superfície considerável da pele e noutras zonas bem mais limitadas — pequenas manchas.

Nunca pudemos afirmar quaisquer sinais de electividade para esta ou aquela região do corpo.

EDEMA.

Este sinal clínico, que chama mais a atenção quer dos familiares quer do médico, é o que mais motiva a consulta e a hospitalização, sendo o mais bem caracterizado do síndroma. Observámo-lo desde o edema discreto maleolar até à infiltração geral de todos os tecidos, como na anasarca.

A grande maioria das vezes está presente nos membros inferiores e sempre simétrico. Outras vezes dá um aspecto característico à face, que aparece como que balofa.

Noutros casos, ainda, verificámos a coexistência de edemas e desidratação. Está hoje provado que estes edemas estão relacionados com os valores muito baixos das proteínas plasmáticas, principalmente da albumina. Não pudemos, em virtude das circunstâncias em que nos encontrávamos, fazer um estudo sobre este aspecto nos nossos casos clínicos, para sua comprovação.

INFILTRAÇÃO GORDUROSA, NECROSE CELULAR E FIBROSE DO FÍGADO.

Não comprovámos o aparecimento destas lesões hepáticas por absoluta impossibilidade em procedermos às punções biópsicas e ainda a exames anátomo e histo-patológicos nos casos de *exitus letalis*.

ALTA MORTALIDADE.

Em 13 casos descritos tivemos apenas 2 casos de morte.

Não fazemos quaisquer considerações por o número de casos ser pequeno. No entanto, todos os autores apresentam estatísticas pessoais ou referências que confirmam realmente a alta mortalidade, mesmo com um tratamento dietético correctamente instituído. Julgamos, no entanto, que esta afirmação só é verdadeira para os casos avançados.

FREQUENTE ASSOCIAÇÃO COM VÁRIAS DERMATOSES.

Verificámos sempre, nos nossos casos, a presença de dermatoses sem características especiais e bem definidas.

Se, porém, algumas crianças apresentavam lesões do tipo pelagróide, pela sua localização e não influenciação pela luz, diferençavam-se das verdadeiras lesões dermatológicas da pelagra.

Verificámos ainda serem as lesões hiperpigmentadas sede frequente de ulcerações e infecções secundárias.

Em dois casos encontrámos lesões de queratinização, que mais tarde originaram zonas de alopecia.

Como alterações vasculares da pele, observámos a relativa frequência com que se formavam úlceras de decúbito, o que nos levou a ter os cuidados especiais nestes casos, para evitar o seu aparecimento.

Cabe-nos aqui fazer uma pequena referência às mucosas.

A mucosa das conjuntivas está o mais das vezes tocada, apresentando frequentemente sinais precursores de queratomalacia; num caso, ulceração da córnea.

PERTURBAÇÕES GASTRINTESTINAIS.

A anorexia é talvez o principal sintoma das perturbações do aparelho digestivo. É por vezes bastante rebelde, o que motiva, por esta razão, a alimentação por sonda. Verificámos que, no entanto, é selectiva, visto que a criança pode, na grande maioria dos casos, alimentar-se como habitualmente.

O sintoma que vem logo a seguir é a diarreia, tantas vezes severa e rebelde no tratamento. Verifica-se sempre na história pregressa que precede a apresentação dos pacientes no hospital.

Geralmente não é bacteriana e liga-se, portanto, à deficiência alimentar.

A infestação parasitária, quase sempre presente em todos os casos, costuma agravá-los; há que atender a este pormenor sempre que, depois da melhoria do estado geral e dos edemas, a diarreia persiste. A atitude terapêutica nestes casos, com as devidas reservas e cuidados, é desparasitar. Assim nos aconteceu em dois casos observados.

IRRITABILIDADE E APATIA MENTAL.

Observámos em quase todos os casos perturbações do psiquismo. As crianças, na sua maioria, apresentavam-se «tontas, apáticas e indiferentes».

As expressões do rosto não podem descrever-se, mas, olhando-as uma vez, não mais esquecem.

Noutros casos observámos, pelo contrário, sinais de irritabilidade, sobretudo aquando do exame médico. No entanto, acabado este, volvem ràpidamente para o marasmo e apatia habituais.

Observações diversas. — Não pudemos deixar de assinalar, sobretudo nas crianças de mais de 2 anos, alterações do aparelho osteomuscular, como sejam deformações do esqueleto, artorpatias e também atrofias musculares.

Verificámos, por último, presença de cárie dentária em bastantes casos.

DESCRIÇÃO CLÍNICA E TERAPÊUTICA INSTITUÍDA

CASO N.º 1. — Matapala, 4 anos, sexo masculino, raça africana. Entrada em 17-7-1956. À entrada: edema generalizado; anorexia pertinaz, apatia mental, cabelos despigmentados, desfrisados e ralos, secura da pele, conjuntivite e descamação, queilite angular, hemorragias gengivais, diarreia; hepatomegalia discreta; altura, 85 cm; peso, 9,25 kg.

Tratamento: leite desnatado, hidrolisatos de proteínas, complexo B.

Evolução boa: desaparecimento da diarreia ao fim de 5 dias e dos edemas ao fim de 29 dias; peso à saída, 10 kg; bom estado geral.

CASO N.º 2. — Tatu, 3 anos, sexo masculino, raça africana. Entrada em 20-8-1956. Cabelos desfrisados, com despigmentação e ralos; certo grau de apatia; lesões pelagróides, anorexia, anemia; parasitose intestinal (oxiúros + ascaris); cárie dentária, diarreia; edema dos membros inferiores e face; altura, 76 cm; peso, 10,125 kg.

Tratamento: leite integral, por não ter aceitado o leite semi-desnatado; depois transição progressiva para uma alimentação própria da sua idade. Extracto hepático com vitamina B_{12}; ferro; complexo B; citrato de piperazina.

Boa evolução, com desaparecimento dos edemas ao fim de 15 dias.

CASO N.º 3 — Joaquina, 19 meses, sexo feminino, raça africana. Entrada em 27-8-1956. Cabelos despigmentados; edema generalizado; diarreia profusa e vómitos e um péssimo estado geral.

Em virtude de o caso ser grave e de urgência, empregámos o plasma na dose de 20 mg por quilograma de peso, repetida diàriamente, bem como toda a medicação indicada nestes casos.

Exitus letalis ao fim de 2 dias.

CASO N.º 4. — Luís Manuel, 2 anos e meio, sexo masculino, raça mista. Entrada em 25-8-1957. Altura, 74 cm; peso, 8,15 kg. Alterações da textura dos cabelos, com despigmentação; zonas de alopecia; estomatite; diarreia e vómitos; edema dos membros inferiores; anemia; anorexia; hiperpigmentação em placas e zonas de descamação; paludismo; parasitismo intestinal (tricocéfalos e oxiúros).

Tratamento: leite semidesnatado+Arobon a 2 %+Nidex; 4 dias depois acrescentámos uma banana durante 5 dias; introdução progressiva de caldo de carne e depois uma alimentação variada e enriquecida em prótidos; frutas de ocasião.

Evolução: a anorexia foi bastante difícil de vencer e, por essa razão, houve que modificar progressivamente o regime, de modo que ele fosse aceite pela criança.

A recuperação de peso foi muito morosa e sempre pouco satisfatória. Os edemas também demoraram a desaparecer (35 dias); peso à saída, 8,135 kg; medicação: ferro, extracto hepático com vitamina B_{12}; Halibut; citrato de piperazina; complexo B.

CASO N.º 5. — Roque Caetano, sexo masculino, 11 meses e meio, raça africana. Entrada em 10-6-1957. Altura, 64 cm; peso, 6,20 kg. Despigmentação dos cabelos; queilite angular; irritabilidade; dermatose das pernas; cárie dentária; pequena hérnia umbilical; helmintíase.

Tratamento: leite semidesnatado.

Evolução: boa, com franca melhoria ao fim de 2 semanas; introdução progressiva da alimentação própria da idade; peso à saída, 6,30 kg.

CASO N.º 6. — Iana, sexo feminino, 14 meses, raça mista; altura, 60 cm; peso, 4,76 kg. Entrada em 15-7-1957. Ascite discreta e edema dos membros inferiores; anorexia; hepatomegalia; cabelos despigmentados; sinal da bandeira; diarreia moderada; manchas de hiperpigmentação por todo o corpo; cárie dentária; helmintíase (ascaris + tricocéfalos).

Tratamento: por não ter aceitado nem o leite desnatado nem o semidesnatado, foi utilizado na dietética inicial o leite integral; depois hidrolisatos de proteínas por via oral; complexo B; preparados de ferro e fígado.

Evolução: ao fim de 25 dias desaparecimento dos edemas; no fim de 44 dias desaparecimento da ascite; melhoria progressiva do estado geral. Cura pelo Resochin; mais tarde citrato de piperazina. Peso à saída, 5 kg.

Caso n.º 7. — Sabudo, sexo masculino, 6 meses, raça africana; altura, 55 cm; peso, 5,85 kg. Entrada em 4-8-1957. Apatia; diarreia; zonas de alopecia, cabelos despigmentados; estomatite; úlcera fagedénica tropical num dos membros inferiores; edema dos pés.

Tratamento: leite semidesnatado, Arobon e Nidex; papa de banana; entrada progressiva no regime próprio da idade ao fim de 25 dias; complexo B; ferro e preparações de extracto hepático. Curado ao fim de 55 dias. Peso à saída, 6 kg.

Caso n.º 8. — Binda, sexo feminino, 4 anos, raça africana. Entrada em 5-9-1957. Grande edema generalizado; olhos quase fechados pelo edema; alteração da textura e despigmentação dos cabelos, que são ralos. Lesões pelagróides da pele; anemia; hemeralopia; queilite angular, glossite, anorexia; irritabilidade; diarreia profusa; helmintíase; ascaris; paludismo; bilharziose vesical.

Em virtude da gravidade do caso, foi feita uma transfusão de heteroplasma, que tolerou bem; analépticos, Percorten, etc.; no entanto, ao fim de 24 horas faleceu em colapso.

Caso n.º 9. — Roque, sexo masculino, 10 anos de idade, raça africana; altura, 1,25 m; peso, 14,500 kg. Entrada em 10-9-1957. Manchas de hiperpigmentação na face, membros superiores e tronco. Despigmentação dos cabelos; edema dos membros inferiores; diarreia e irritabilidade; helmintíase (oxiúros + ancilóstomos).

Tratamento: leite integral com papa de banana; ao fim de 15 dias, regime dietético próprio da idade; frutas, legumes verdes, leite; suplemento vitamínico; preparações de ferro e extracto hepático; complexo B. Intercorrência com febre palustre, curada pelo Aralen. Tratamento da sua helmintíase com tetracloreto de carbono e óleo de quenopódio. Os edemas desapareceram ao fim de 20 dias. Curado ao fim de 65 dias. Peso à saída, 14,350 kg.

Caso n.º 10. — Tariga, sexo feminino, 8 anos, raça africana; altura, 1,16 m; peso, 20,500 kg. Entrada em 20-10-1957. Apatia mental, por vezes irritabilidade; edema dos pés e maléolos; lesões pelagróides; despigmentação dos cabelos; esplenomegalia; bilharziose vesical; sinais de avitaminose A.

Tratamento: repugnância total por qualquer leite; hidrolisatos de proteínas, por via oral; regime dietético constituído por uma alimentação variada e rica em proteínas. Antianémicos: complexo B; Halibut; Nilodin.

Evolução: boa recuperação, funções digestivas normais ao fim de 5 dias. Edemas desaparecidos ao fim de 8 dias. Melhoria progressiva do estado geral. Alta ao fim de 65 dias. Peso à saída, 14,650 kg.

CASO N.º 11. — Babu, sexo masculino, 3 anos, raça mista; altura, 71 cm; peso, 7,10 kg. Entrada em 19-11-1957. Aspecto miserável; apatia; edema generalizado; anemia; esplenepatomegalia; despigmentação dos cabelos; estomatite; zonas hiperpigmentadas por todo o corpo; helmintíase; otite; conjuntivite; úlcera fagedénica da perna; grande hérnia umbilical; cárie dentária; vómitos e diarreia.

Tratamento: heteroplasma no primeiro dia; depois pequenas doses de leite desnatado, que foi preciso dar por sonda e em pequenas quantidades; no entanto, como a diarreia persistia, diminuímos a concentração do leite, ao qual acrescentámos um cozimento de farinha de arroz. Como a diarreia persistisse ainda, e começasse a ter intolerância para o leite desnatado, instituímos o Pelargon; mais tarde passámos ao Eledon e papa de banana com bolacha. Medicação subsequente: ferro; extractos hepáticos com vitamina B_{12} altamente concentrada; Bécozyme. A diarreia foi desaparecendo e, quando os edemas também desapareceram (ao fim de 33 dias), instituímos um regime rico em proteínas, constituído por caldo de carne, fígado de vitela, frutas, legumes verdes, além de pão, batata, arroz, etc.

A criança reagiu bem ao tratamento e abandonou a enfermaria, clìnicamente curada, ao fim de 90 dias de tratamento.

Com a devida oportunidade, fizemos cura pelo Aralen e citrato de piperazina.

CASO N.º 12. — Dominga, sexo feminino, 5 meses de idade, raça africana; altura, 53 cm; peso, 3,750 kg. Entrada em 21-11-1957. Despigmentação dos cabelos; lesões pelagróides; apatia; edema dos membros inferiores; anorexia e diarreia; otite média aguda.

Tratamento: continua a alimentar-se ao seio da mãe; alimentação complementar com leite semidesnatado; penicilina durante 4 dias;

dificuldade para tomar o leite semidesnatado; procura elevar-se a secreção láctea da mãe, para o que lhe demos um regime alimentar completo e variado. Por não aceitação do leite desnatado depois de muita insistência, fornecemos-lhe Pelargon, que com relutância sempre vai aceitando; mais tarde alimentação exclusiva de leite materno; suplementos vitamínicos. Recupera progressivamente o peso; introdução de farinha Maizena. Continua a melhorar o estado geral e a recuperar o peso. Sai do hospital, ao fim de 60 dias, com o peso de 4,780 kg e desaparecimento de toda a sintomatologia de *kwashiorkor*.

CASO N.º 13. — António, sexo masculino, 5 meses de idade, raça africana; altura, 59 cm; peso, 3,800 kg. Entrada em 10-12-1957. Despigmentação dos cabelos; edema dos membros inferiores; esplenomegalia; lesões de hiperpigmentação; vómitos e diarreia.

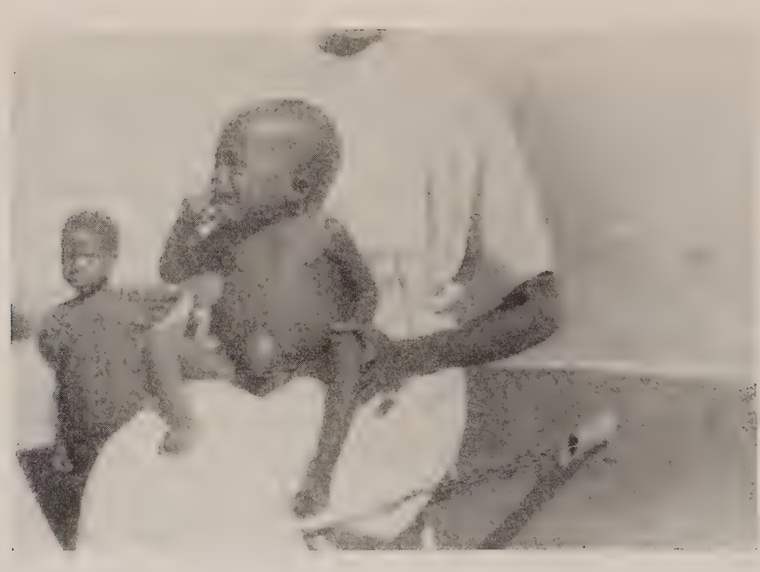
Tratamento: pausa hídrica durante 6 horas; recomeço com a alimentação ao seio da mãe; mamada insuficiente por hipogalactia; recusa de qualquer outro leite; algum tempo depois a mãe, pela boa alimentação que teve, já o pode alimentar convenientemente; suplementos vitamínicos; começa lentamente a recuperar o peso e a melhorar.

Sai curado ao fim de 45 dias, com o peso de 4,250 kg.

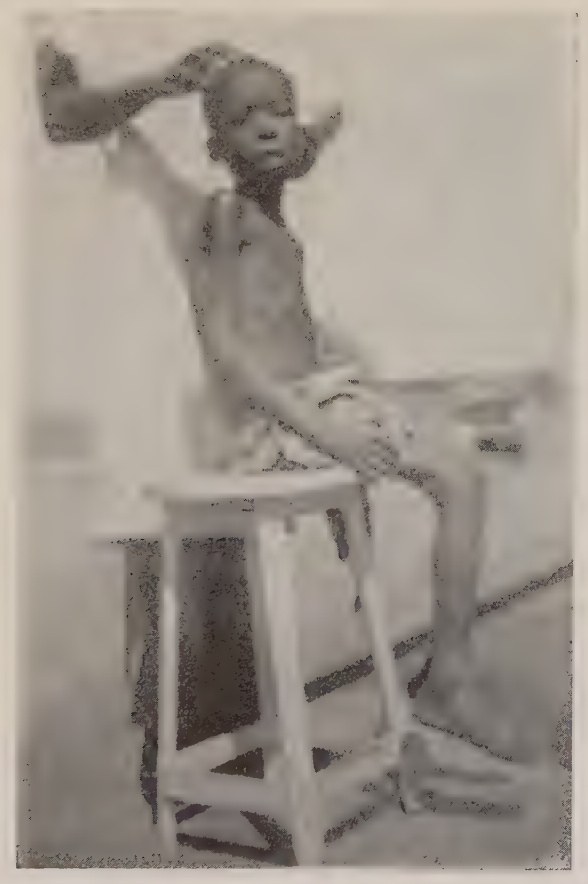
ETIOPATOGENIA DO *KWASHIORKOR*

Na exposição que no capítulo anterior fizemos das condições de vida alimentar das crianças nativas da região onde encontrámos esta enfermidade observámos todas as causas que originam o referido síndroma.

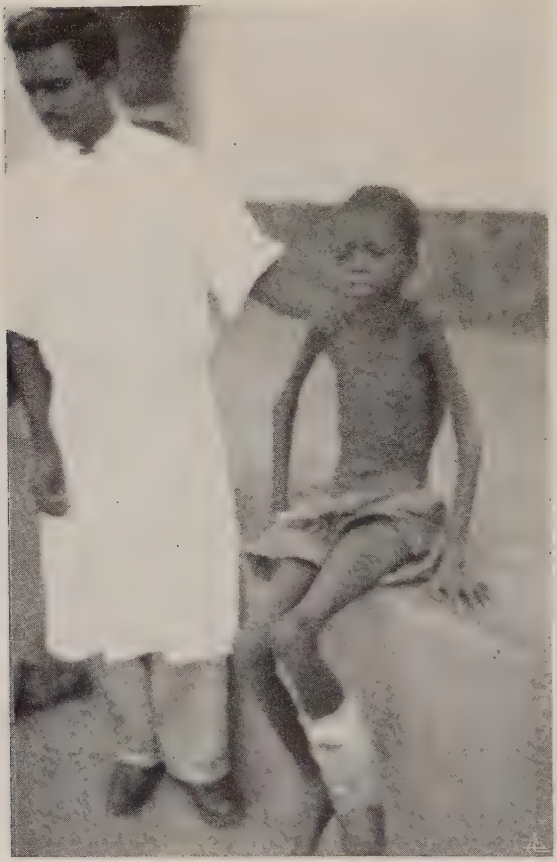
Assim, nas histórias clínicas dos casos apontados, verificámos que a maior parte delas, embora nascidas a termo, pareciam prematuras. Além do mais, ainda observámos, pelos dados fornecidos pelo dispensário pré-natal, que a maioria das mulheres grávidas que o frequentavam eram possuidoras de anemias filiadas geralmente na deficiência do seu regime alimentar, desfalcado qualitativa e quantitativamente, e, ainda, de parasitoses intestinais e bilharziose vesical, factores estes também condicionantes de um estado biológico muito



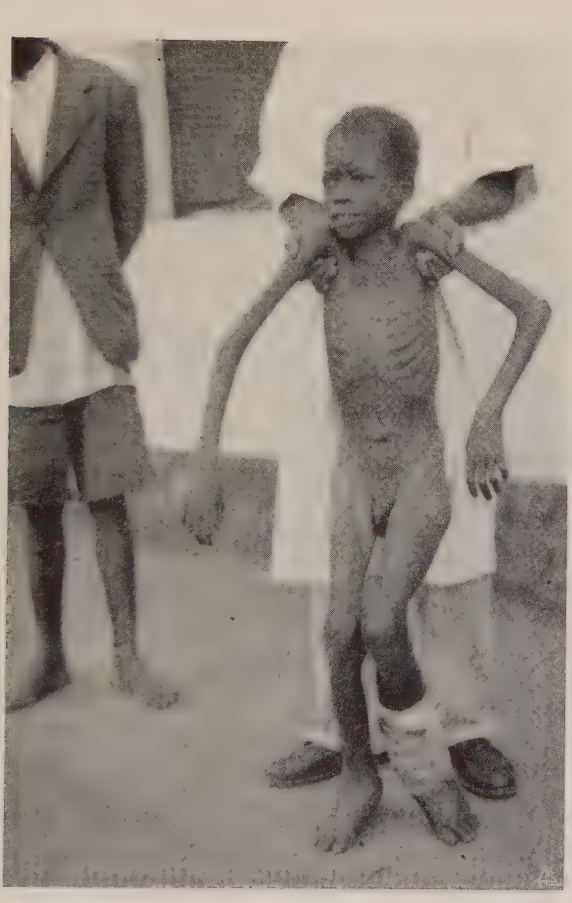
Caso n.º 4 — Notar os edemas dos membros inferiores, o avultamento do abdome e a despigmentação dos cabelos



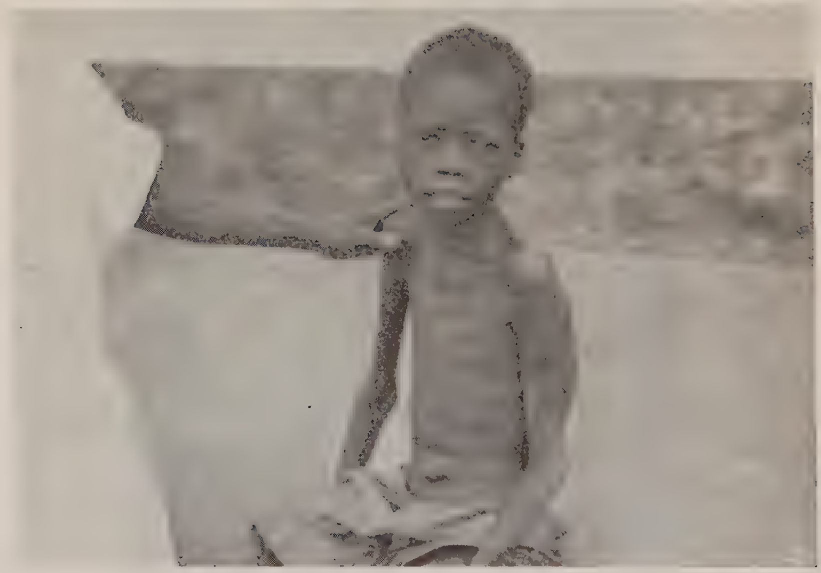
Caso n.º 10 — Notar o edema dos membros inferiores, a despigmentação dos cabelos e a dermatose nos membros inferiores



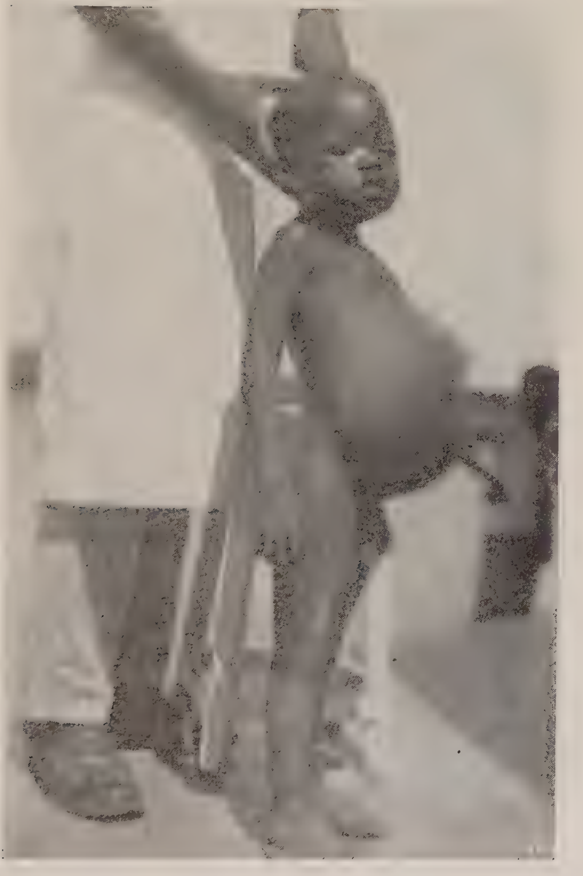
Caso n.º 9 — Notar o edema dos membros inferiores, a despigmentação dos cabelos, a apatia e ainda a atrofia dos membros inferiores



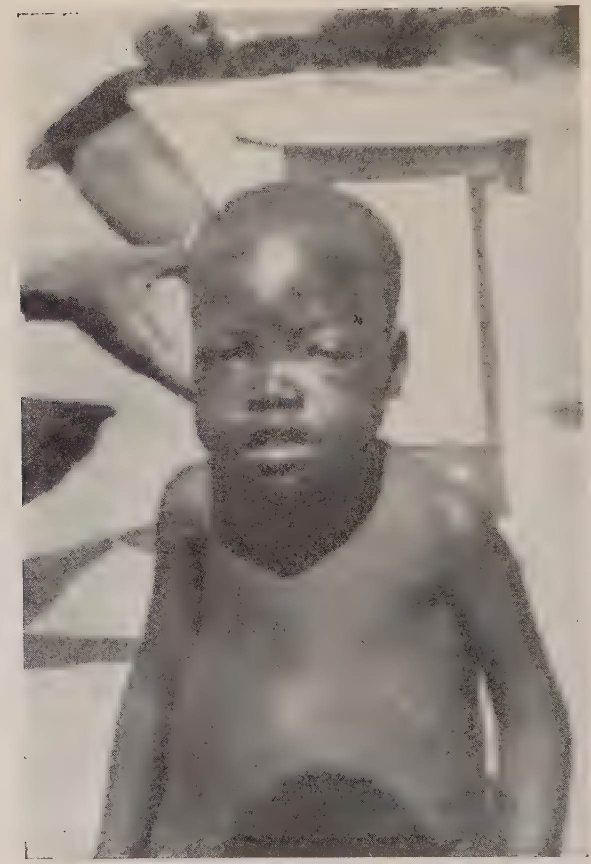
Caso n.º 9 — Notar a ancilose das articulações do joelho por imobilidade



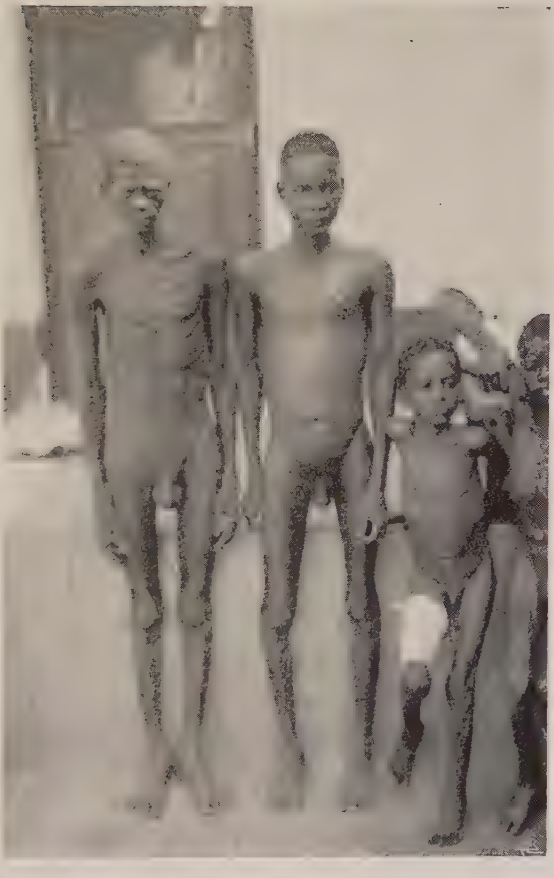
Caso n.º 9 — Notar a expressão miserável



Caso n.° 1 — Notar a elevação do ventre, o edema das pernas e do pénis, o edema da face e lesões cutâneas do membro inferior direito



Caso n.º 1 — Notar o edema da face e olho esquerdo ligeiramente fechado pelo edema das pálpebras; despigmentação e modificação da textura dos cabelos



Irmão e pai do caso n.º 1 — Notar o aspecto de desnutrição de ambos precário e que originam, como se sabe, prematuridade e debilidade congénitas nos nascituros. Além disso, as mães das crianças, nos casos observados, referiram quase sempre agalactia ou hipogalactia.

A incidência das idades de aparecimento do *kwashiorkor* começou depois do primeiro semestre de vida, altura em que a criança nativa passa, sem qualquer transição, para o regime alimentar do adulto, já de si tão deficiente, tendo-se verificado imediatamente uma paragem do seu peso e estatura.

Referimos ainda ser esta a idade em que a criança começa a ser atingida pelas diversas parasitoses intestinais e pelo paludismo, que, se consegue sobreviver, terá de acrescentar à sua já bem pesada existência.

Notámos a influência da época estacional — húmida, quente e chuvosa — no aparecimento dos casos, ao mesmo tempo com as diarreias estivais.

BIOQUÍMICA

As aquisições modernas no domínio da bioquímica, o conhecimento cada vez mais aprofundado do metabolismo dos proteicos, lipidos, glucidos e vitamina e sua respectiva correlação e ainda o conhecimento mais perfeito da histopatologia do fígado, pâncreas e glândulas secretoras intestinais, sobretudo nos casos estudados em vida e, por conseguinte, menos evoluídos, pela punção — biopsia do fígado —, tornaram possível um melhor conhecimento do mecanismo íntimo e desencadeante deste síndroma que hoje nos ocupa.

A interpretação científica deste síndroma, tão proteiforme sobretudo nas suas fases iniciais, tem, pelos motivos expostos, avançado muito com estas modernas aquisições do seu estudo.

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A interpretação da capacidade funcional do tubo digestivo pela medida de absorção intestinal das proteínas e gorduras é um processo semiológico de alto valor e mesmo de segurança para o clínico e conduz a resultados notáveis, no que diz respeito não só ao diagnóstico mais precoce, como ainda a seguir-se uma mais pormenorizada evolução do processo em relação à terapêutica instituída.

O conhecimento de que o *kwashiorkor* é um síndroma originado pela carência das proteínas nos regimes alimentares, quer nos povos subdesenvolvidos, quer ainda naqueles que vivem em precárias condições económico-sociais em outros continentes, está actualmente na ordem do dia em todas as questões no domínio da nutrição em geral.

Na realidade, o conhecimento actual deste problema de malnutrição, que se embrica sucessivamente em todos os conhecimentos do metabolismo em geral, tem prendido a atenção de numerosos cientistas, nos diversos departamentos da ciência médica que têm estudado esta apaixonante questão.

E, assim, aos conhecimentos empíricos do início, desde que esta enfermidade foi descrita pela primeira vez, em 1926, por Parker e mais tarde mais evidenciada por Cecily Williams (1935) até aos estudos anátomo-patológicos bem documentados de Gillman — que em 1934 descreveu pela primeira vez, com toda a exactidão, as lesões hepáticas (infiltração gorda) —, sucederam-se várias outras tentativas de aprofundamento anátomo-patológico, de que se destacam os estudos de Davies (1948) e Veghelyn (1950), os quais pela primeira vez assinalaram no pâncreas lesões constituídas pelas células acinares e verificaram ainda que estas lesões precediam as hepáticas.

Além disto, assinalaram mais tarde uma atrofia das células secretoras do intestino.

Devido ao conhecimento mais exacto, portanto, da anátomo-patologia do tubo digestivo e suas glândulas anexas, pôde estudar-se melhor a sua fisiopatologia e, deste modo, Dean e Schwartz chegaram à conclusão de que «há uma falência da síntese proteica e insuficiência enzimática generalizada».

Por aqui se verifica haver, pela falta ou carência de proteínas nos regimes alimentares, uma redução no *apport* proteico plasmático e ainda, pela redução da actividade enzimática, um dismetabolismo azotado.

E assim se compreende que os alimentos não sejam totalmente digeridos nestas partes do tracto intestinal, dando como possível consequência uma anorexia o mais das vezes pertinaz e outras perturbações digestivas, que sempre verificámos nos casos que tivemos ocasião de estudar.

Pelos estudos feitos por K. Holemans e A. Lambrechts, no Congo Belga, estes autores verificaram que há uma anomalia no comportamento do metabolismo dos lipidos, por uma perda do fósforo lipídico pelas matérias fecais, e relacionaram-na com o desaparecimento duma substância lipotrópica que influenciava a integridade histológica do fígado por sobrecarga gorda.

TRATAMENTO

REGIME DIETÉTICO.

De entre os tratamentos que têm sido preconizados para este síndroma, os últimos trabalhos científicos que dele se têm ocupado dão a preferência ao regime dietético constituído pelo leite desnatado, semidesnatado e até muitas vezes o leite integral.

O tratamento-base, portanto, são as proteínas do leite, conjuntamente com uma dieta variada e adequada em relação com a idade e estado geral da criança.

No entanto, na prática corrente verifica-se ser necessário atender à tolerância da criança, sendo por vezes de aconselhar a mudança de um regime teòricamente mais indicado para um outro inferior em qualidade, mas que a criança tolere melhor. Neste ponto verificámos, por experiência pessoal, que o tratamento dietético tem de ser prudente e progressivo, não só pela adaptação que a criança tem de fazer ao regime que se empregar, como ainda para vencer a anorexia, que em muitos casos é difícil de debelar.

E, assim, tendo em vista o que atrás expomos, adoptámos o critério de Galopan, que começa por dar 33 g de leite desnatado, subindo gradualmente até atingir 60 g, com o que obteve resultados muito satisfatórios.

Embora o cálculo teórico das proteínas animais a instituir nestes regimes dietéticos seja de, pelo menos, 4 g até 7 g por quilo de peso corporal, verificámos, como acima dissemos, ser de início, nos casos observados, absolutamente impraticável por intolerância. Em todas as crianças internadas demos, pois, preferência ao leite desnatado ou semidesnatado, segundo o critério de Galopan. Num caso, porém, usámos o leite integral empregado na diluição conveniente, por absoluta impossibilidade de aceitação quer do leite desnatado quer do semidesnatado.

Noutro caso ainda conseguimos vencer a anorexia seguindo um esquema de Trowel baseado na sua experiência no Hospital de Kampala, e que foi o seguinte:

a) 400 g de banana esmagada;
b) 200 g de leite em meio litro de água.

Em dois casos de *kwashiorkor* em que as crianças eram apenas alimentadas ao seio mantivemos este tipo de alimentação, aumentando a secreção láctea das mães, que era insuficiente, por uma melhor alimentação e por uma medicação tónica geral.

Quanto às crianças maiores, entre os 2 e os 4 anos, passámo-las progressivamente para um regime mais completo e variado, introduzindo na sua alimentação carnes, fígado, legumes verdes e frutas, e mais tarde, quando a digestão era boa, passámos ao regime normal, ajuntando como complementos preparações antianémicas, vitaminas, agentes lipotrópicos e tratamento anti-infeccioso e parasitário, quando necessários.

Como critério de apreciação, baseámo-nos apenas, por dificuldades técnicas, nas modificações do aspecto geral que nos permitissem, por outro lado, um estudo paralelo da bioquímica do sangue dos nossos doentes e também um estudo da capacidade funcional do tubo digestivo, para a medida da absorção intestinal das proteínas e gorduras pelo método directo dos *bilans*.

A apreciação da marcha e da evolução do síndroma, repetimos, foi feita pela apreciação dos seguintes dados clínicos:

1) Modificação da curva do peso;
2) Aspecto geral;
3) Desaparecimento das perturbações digestivas.
4) Desaparecimento dos edemas.

COMPLEMENTOS AO REGIME DIETÉTICO

VITAMINAS.

Há grande controvérsia sobre o emprego de vitaminas neste síndroma. Parece, no entanto, que, não devendo ser empregadas como terapêutica curativa, como ao princípio se julgava, no início do tratamento, elas devem entrar como complemento do regime dietético numa fase mais tardia do tratamento, já quando há necessidade de instituir um regime normal, para que fiquem asseguradas todas as necessidades do organismo.

A preferência, nestes casos, vai umas vezes para os factores do complexo B e outras vezes, quando custem a desaparecer os sinais clínicos de avitaminose A, para a vitamina A.

AGENTES LIPOTRÓPICOS.

Empregámo-los em alguns dos casos, por sabermos que, muitas vezes, antes do *kwashiorkor* já as crianças haviam tido doenças tropicais que decerto lhes atingiram o fígado, para o qual esta terapêutica é necessária e comprovadamente eficaz, como bilharzioses, paludismo, etc.

PREPARAÇÕES ANTIANÉMICAS.

Pelo facto de a grande maioria dos indígenas desta região terem com frequência anemias ferroprivas e macrocíticas e ainda de haver sinais clínicos de anemia, empregámos quer a terapêutica marcial, como adjuvante do tratamento geral e ainda porque alguns dos nossos pacientes tinham concomitantemente ancilostomíase, quer a terapêutica pelo extracto hepático com vitamina B_{12}.

OUTRAS TERAPÊUTICAS ASSOCIADAS

Aplicámos num caso sòmente, em virtude da sua gravidade, o plasma heterólogo, em virtude da urgência e para lutar contra a hipoproteinemia e desidratação, em virtude de uma diarreia profusa, datando de há algum tempo e que uns três dias antes de o doente ter vindo à consulta se tinha exacerbado. Verificámos uma resposta bôa e imediata à terapêutica instituída, pela melhoria rápida do aspecto geral.

TRATAMENTO ANTI-INFECCIOSO.

A maioria dos casos tratados tinham associados, quer parasitoses específicas, como o paludismo, quer parasitismo intestinal em maior ou menor grau.

Recorremos no primeiro caso, para o seu tratamento, aos medicamentos menos tóxicos e em doses prudentes; e no segunde caso só iniciámos a terapêutica depois de a criança ter recuperado e estar em condições de poder sem perigo suportá-la.

RESULTADOS E CONCLUSÕES

Dos treze casos que apresentámos e que tivemos ocasião de tratar, instituímos em todos um regime dietético apropriado e adequado, tendo em vista não só as necessidades calóricas e quantitativas, mas ainda uma correlação adequada entre os seus vários componentes, de modo que correspondessem à capacidade digestiva nos vários períodos de desenvolvimento das crianças que tivemos ocasião de tratar, tendo sofrido apenas dois insucessos.

Estes dois últimos casos, porém, apareceram à consulta quase em estado pré-mortal, que com as nossas disponibilidades, quer terapêuticas quer ainda laboratoriais, não foi possível recuperar.

Todos os outros casos tiveram êxito, porém.

Além do leite desnatado ou semidesnatado, empregámos produtos de origem local (carne, peixe, frutas, farinha, mandioca), respeitando o gosto dos doentes.

A duração do internamento foi, no entanto, mais longa do que a que a mairia dos autores refere, simplesmente com a finalidade de os manter o mais tempo possível afastados do meio familiar, que, pela sua pobreza e ignorância, os poderiam novamente conduzir à situação anterior.

RESUMO

Foram observados pela primeira vez alguns casos de *kwashiorkor* de formas muito severas e bastantes casos de formas mais atenuadas, na circunscrição de Mutarara, província de Moçambique, o que permitiu dar-se notícia e fazer-se um estudo pormenorizado do ponto de vista clínico.

Aproveitámos elementos fornecidos pelo dispensário daquela localidade, colhidos durante quatro anos de observação constante das crianças nativas que o frequentaram.

Pôde fazer-se uma seriação dos casos apresentados e a sua devida selecção, mercê de circunstâncias meramente ocasionais, relacionadas com o modo de viver e hábitos de vários grupos de populações primitivas.

Relacionou-se ainda o aparecimento desta policarência com epidemias de fome durante os anos desta ocorrência e fez-se um estudo pormenorizado do ponto de vista económico-biológico, verificando-se sempre um estado muito precário daquelas duas condições, tradução evidente da pobreza do regime alimentar, quer em proteínas animais, quer em proteínas vegetais, e ainda de uma exiguidade bem marcada de princípios vitamínicos.

Fez-se um estudo comparativo e estatístico sobre o peso e altura de crianças normais em relação com aquelas que se apresentavam com o *kwashiorkor*.

Apurámos, por diversos inquéritos, as condições de vida alimentar e hábitos tradicionais e familiares, quer da população em geral, quer das mulheres grávidas, quer também das mulheres que amamentam, e ainda os métodos de alimentação das crianças nas diversas idades.

Iniciámos o tratamento dos casos apresentados sob o plano essencialmente dietético, não sem atendermos sempre ao estado geral dos casos observados, às suas perturbações digestivas e ao condicionalismo dos hábitos alimentares.

Todos os tratamentos foram postos, não em fórmulas rígidas e estandardizadas, mas sim por tacteamento da susceptibilidade individual de cada caso; não menosprezámos o valor da vitaminoterapia; nos casos de concomitante associação com parasitoses intestinais, paludismo e outras tivemos os cuidados habituais na dose e na oportunidade da respectiva medicação.

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THE CAUSES OF THE PROTEIN DEFICIENCY IN A TROPICAL REGION: PERNAMBUCO, BRAZIL

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The state of Pernambuco is one of the most important in Brazil and its capital, Recife, is the third city in Brazil. The state may be divided according to the soil, climate and plants in the forest area, and the caatinga area; the last one is a dry and semi-deserted region.

The problem of protein deficiency was discussed from a physiological point of view and great differences of opinions were shown concerning the minimal and medium intake of protein in man and experimental animals. Supporting this opinion several ones were cited, such as the following: Voit said that the protein per capita a day will be 118 g; Chittenden said that it is 44 g to 53 g; Sherman said that it is 44 g; Mc Lister thinks that the efficiency of American soldiers in the last war was due to a high intake of protein (180 g a day). According to Houssay, the Argentine peasant eats 2 kg or 3 kg of meat every day. The British Ministry of Health advises 50 g of first class protein a day (meat, eggs, milk).

Discussing these different opinions, it was shown the effect of some factors, such as mineral elements, vitamins, B_{21} and B_6, carbohydrates on the protein metabolism and the nitrogen fixation.

Some experimental works made in the Institute, including the chromatographic analyses of beans *(Phaseolus vulgaris* and *Vigna sinensis)*, were discussed, and it was pointed out the value of the *Vigna sinensis* in nutrition.

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The inquiries on the protein deficiency in this region were also discussed.

Afterwards it was studied the cause of the protein malnutrition in this region.

The acidity of the tropical soil and the destruction of the forests in the region discussed were pointed out. It is a general practice in the tropical countries, where the trees are used like energetic material. This general use of wood energy in many countries leads to the weakness of the soils. Examples of the decay of civilizations were given and was shown the great and fundamental importance of the carbone and nitrogen cicles in «live world». It was pointed out the connection between the soil, plants and animals and the importance of the soil on the development of any civilization.

SUBSECTION 3

ANAEMIAS AND ABNORMAL HAEMOGLOBINS

1ST SESSION: *Friday, September 5th — 4:30 to 6:00 p.m. Room A*

The meeting opened under the chairmanship of Dr. A. Dubois. The Rapporteurs were Dr. A. W. Woodruff and Dr. Carlos P. Trincão, and the Secretary was Dr. Egídio R. R. Gouveia.

The following papers were presented and discussed.

HEMOGLOBINAS ANORMAIS: PROPRIEDADES E MÉTODOS DE IDENTIFICAÇÃO

CARLOS TRINCÃO

Embora as hemoglobinas do homem e da maioria dos vertebrados tenham igual peso molecular, as mesmas quatro ferroprotoporfirinas e igual capacidade de fixar quatro volumes de oxigénio por molécula, diferem pelas respectivas globinas que lhes conferem especificidade.

Em 1866 Körber demonstrou, dentro da mesma espécie animal, diferenças na desnaturação pelos alcalis das hemoglobinas fetal e adulta, revelando-se a primeira mais resistente a este tratamento químico. Só em 1910 Walkulenko verificou o mesmo facto relativamente às hemoglobinas dos sangues do cordão umbilical do recém-nascido e do homem adulto. Mais recentemente, Vecchio (1946) pôde afirmar a presença de hemoglobina de tipo fetal no sangue dos indivíduos com *thalassaemia major*.

Averiguou-se ainda que o sangue do adulto normal contém uma pequena percentagem de hemoglobina fetal, que pode aumentar em circunstâncias várias: anemia perniciosa progressiva, anemias crónicas arregenerativas, anemias consecutivas a tumores, na gravidez molar

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e, principalmente, nos doentes de anemia de células falciformes ou de talassodrepanocitose.

Em 1949 Pauling e col., sujeitando à electroforese a hemoglobina de doentes de anemia de células falciformes e vectores assintomáticos de drepanocitose, provaram depender esta anomalia da presença, nos glóbulos rubros, duma hemoglobina diferente da hemoglobina adulta normal.

De então para cá têm-se isolado numerosas outras variedades patológicas de hemoglobina, especialmente nas raças que habitam as regiões inter e subtropicais.

Em seguida à descoberta de Pauling e col., começou a designar-se por A a hemoglobina do adulto normal, por F a hemoglobina fetal e por S a hemoglobina da drepanocitose *(sickle-cell anaemia)*. Em Janeiro de 1953 o United States Public Health Service promoveu uma reunião dos investigadores interessados no assunto, na qual se resolveu modificar esta nomenclatura, passando a designar-se pela letra B a hemoglobina falciforme e pelas outras letras do alfabeto, de C a E e de G em diante, as variedades de hemoglobina já descobertas, ou que de futuro se viessem a identificar. As letras A e F continuaram a designar a hemoglobina normal do adulto e a hemoglobina fetal. A força do uso fez com que a letra S tenha continuado a atribuir-se à hemoglobina falciforme, perdendo-se assim a letra B para a classificação das hemoglobinas.

Até hoje isolaram-se as seguintes hemoglobinas anormais: C, D, E, G, H, I, J, L, M, N, O (Buginese X), P (Galveston) e Q, e as variedades Fessas e Papaspyrou, encontradas transitòriamente no sangue dum recém-nascido, e Bart's. Além destas, mais outras variedades se descreveram, mas sem indicação de características que permitam a sua nítida individualização. Estão nestas condições a hemoglobina anormal mencionada por André e col. e aquela a que mais recentemente Chernoff e col. atribuíram a letra R.

Referiremos os vários métodos empregados para o seu isolamento e caracterização.

Cristalografia. — Enquanto a oxi-hemoglobina A cristaliza formando prismas ortorrômbicos terminados por faces bipiramidais, a hemoglobina F cristaliza no sistema triclínico.

Nos solutos de hemoglobina S expostos a baixa tensão de oxigénio, e seguidamente observados em microscópio de contraste de fase, formam-se tactóides, isto é, corpos alongados resultantes da agregação ou polimerização de moléculas, não tão organizados como cristais, mas ainda capazes de birrefringência à luz polarizada e que se assemelham aos eritrócitos em transformação falciforme.

Espectro de absorção na luz visível. — O espectro de absorção da hemoglobina é dado pela curva de qualquer das suas constantes fotométricas (geralmente o coeficiente de extinção específica) nos vários comprimentos de onda da luz incidente.

As curvas de absorção da luz das várias hemoglobinas são idênticas à da hemoglobina A, com excepção da hemoglobina M, que mostra o espectro da meta-hemoglobina.

Espectro de absorção no ultravioleta. — Os espectros da absorção das hemoglobinas A e F diferem porque, enquanto a primeira tem uma zona de absorção, na faixa da triptofana, a 2910 Å, a segunda tem a zona correspondente a 2890 Å.

As restantes hemoglobinas têm um espectro de absorção no ultravioleta semelhante ao da hemoglobina A, com excepção da variedade Bart's, cujo espectro de absorção no ultravioleta se assemelha ao da hemoglobina F.

Espectro de absorção no infravermelho. — Penati e col. compararam os espectros de absorção das hemoglobinas A e F na zona infravermelha, entre 2 μ e 11 μ, observando 3 faixas de absorção, uma a cerca de 3 μ, que tem origem nas vibrações de valência do hidrogénio em relação com os átomos de carbono e azote, outra a 6,07 μ e 6,52 μ, seguidas duma faixa de pequena intensidade, em especial a 7,7 μ. Em duas faixas, a 7,7 μ e a 8,03 μ, a densidade óptica é maior para a hemoglobina fetal que para a hemoglobina A, diferença explicável por diversa disposição das cadeias polipeptídicas.

Solubilidade. — Uma proteína, ou uma mistura de proteínas, sem interacção entre si dissolve-se até saturação do solvente para cada um dos componentes. Se entre as várias proteínas houver interacção, a concentração do soluto, em relação à quantidade de proteína adicionada, exprime-se por uma curva. Portanto, a comparação da solubilidade de misturas diferentes só é possível quando se mantenha constante a relação da quantidade total da proteína para a do solvente.

Itano idealizou uma prova que tem o mérito de distinguir as hemoglobinas S e D que a electroforese em papel não consegue separar, baseada na diferença das respectivas solubilidades. Consiste em estudar a dissolução de 50 mg do pigmento, reduzido pelo metabissulfito de sódio, em dois solutos de fosfatos mono e dipotássico, com as concentrações 2,24 M e 2,58 M. Quando não haja precipitação no primeiro soluto, tenta-se a dissolução de igual quantidade de hemoglobina no segundo.

Os homozigotos SS e os heterozigotos SD têm uma solubilidade da hemoglobina reduzida no tampão de fosfatos 2,24 M, inferior a 5 g por litro.

O quadro projectado mostra as solubilidades das diferentes variedades de hemoglobina nesta prova.

A baixa de solubilidade característica da hemoglobina S apenas se encontra na sua forma reduzida.

A hemoglobina H é uma proteína instável, pois precipita irreversìvelmente nos seus solutos.

Afinidade para o oxigénio. — A hemoglobina F tem menos afinidade para o oxigénio que a hemoglobina A, ficando a curva de dissociação do oxigénio dissolvido na primeira à direita da da segunda. Também na anemia de células falciformes a curva de dissociação do oxigénio está desviada para a direita, mas nos heterozigotos AS a mesma curva é normal.

De há muito se sabe que as baixas de tensão do oxigénio provocam a transformação falciforme dos eritrócitos que contêm hemoglobina S, mais ràpidamente nos homozigotos que nos heterozigotos. Várias técnicas existem para evidenciar este fenómeno, desde a simples observação do sangue, em preparação selada, entre lâmina e lamela, eventualmente precedida de estase no território vascular onde o sangue vai ser colhido, à anoxia provocada pela junção de bactérias ou fungos fermentadores da glicose, à simples junção de substâncias redutoras, ou à passagem duma corrente de anidrido carbónico. Mesmo os processos mais modernos podem dar resultados negativos em presença de pequenas quantidades de hemoglobina S. Por outro lado, podem verificar-se, em circunstâncias ainda obscuras, pseudofalciformações que têm levado à suposição da existência de focos extraordinàriamente elevados de drepanocitose em populações onde, mais tarde, o método electroforético as não confirmou.

Provas de desnaturação. — Colocada em meio fortemente alcalino, a hemoglobina dissocia-se em heme e globina. O primeiro oxida-se, originando a hematina; a globina desnatura-se, ficando depois a hematina, com a qual forma a cat-hemoglobina, cujo espectro de absorção é diferente do da hemoglobina.

Para que este tipo de prova seja aplicável a uma mistura de hemoglobinas é preciso que: 1.º, a reacção de desnaturação se verifique em relação às concentrações das hemoglobinas presentes; 2.º, os espectros de absorção da proteína nativa e desnaturada sejam os mesmos para ambos os componentes; 3.º, a perda de solubilidade e a alteração dos espectros decorram simultâneamente; 4.º, a fase solúvel apenas contenha hemoglobina desnaturada; 5.º, durante a reacção o pH permaneça constante.

Estas condições verificam-se para misturas de hemoglobina adulta e fetal, embora os doseamentos fotométricos e por precipitação não sejam coincidentes, provàvelmente porque as propriedades em que se baseiam não variam sincrònicamente.

Há perto de um século que se conhece a resistência da hemoglobina fetal à desnaturação pelos alcalis. Das várias hemoglobinas anormais, apenas a hemoglobina Bart's é resistente à desnaturação alcalina.

A hemoglobina fetal não só é resistente à desnaturação alcalina, como também o é à desnaturação pelos ácidos.

Composição em aminácidos das globinas. — São nítidas as diferenças entre as hemoglobinas A e F, por a segunda conter maior quantidade de treonina, serina, metionina e talvez também de ácido glutâmico, mas principalmente de isoleucina (que se não encontra nas hemoglobinas A, S e C depuradas electroforèticamente). Nota-se ainda a sua menor riqueza em prolina, valina, tirosina, histidina e talvez alanina.

A hemoglobina C contém maior quantidade de lisina que a A.

A hemoglobina H tem, como a hemoglobina F, maior percentagem de leucina e menor de prolina, tirosina, valina e histidina; todavia, pelo seu conteúdo de serina e treonina, aproxima-se da hemoglobina A.

Ingram digeriu pela tripsina a globina das hemoglobinas A, S e C, de maneira a separá-las em pequenos peptidos, que depois sujeitou a electroforese e a cromatografia em papel. Das suas interessantes experiências concluiu que as hemoglobinas S e C diferem da A porque, em determinada cadeia peptídica onde aquela tem uma molécula de ácido glutâmico, tem a hemoglobina S uma molécula de valina, a hemoglobina C uma molécula de lisina, etc.

Grupos N-terminais. — Porter e Sanger demonstraram que os grupos N-terminais das cadeias polipeptídicas pertencem à valina. A hemoglobina A tem 4 ou 5 grupos N-terminais valínicos e a hemoglobina F apenas 2,6. As hemoglobinas S, C e E não diferem da A sob este aspecto.

Grupos C-terminais. — São em número idêntico nas hemoglobinas A, S, C e F. Uma vez que nas hemoglobinas A, S e C o número de grupos C-terminais não concorda com o número de grupos N-terminais, é provável que nelas um ou mais dos primeiros grupos não estejam em posição de reagir com a carboxipeptidase. Na hemoglobina F os grupos C- e N-terminais estão ligados entre si, sugerindo a existência de duas cadeias polipeptídicas distintas.

Grupos SH. — Hommes e col. verificaram que as hemoglobinas A, S e C contêm 8 meias moléculas de cistina por molécula de hemoglobina, enquanto a hemoglobina F apenas tem 6, donde concluem que as três primeiras hemoglobinas devem ter duplas ligações S-S, ao passo que na hemoglobina F é provável que 2 meios resíduos de cistina estejam ligados por uma ponte dissulfidrílica.

Imunologia. — Apenas a hemoglobina fetal tem propriedades antigénicas que permitem a preparação de anti-soros que a precipitam. Os anti-soros que se têm preparado a partir de sangues de homozigotos SS ou CC foram obtidos à custa da pequena fracção de hemoglobina fetal contida neles, como é demonstrado pelas suas reacções.

Cromatografia. — Sansone e Cusmano separaram por cromatografia ascendente em papel a hemoglobina adulta da fetal. O método foi usado por outros autores, mas é principalmente à cromatografia em coluna que se devem resultados importantes. Já Boardman e Partridge tinham assim separado as hemoglobinas A e F, mas coube a Huisman e col. o aperfeiçoamento do método. Os autores fazem a cromatografia numa resina trocadora de catiões, a amberlite I R C 50, com a qual ràpidamente conseguem separar as várias hemoglobinas existentes numa mistura e dosear pequenas fracções destas.

As várias hemoglobinas até agora experimentadas dispõem-se nos cromatogramas, pela ordem perfeitamente conhecida.

Mais recentemente, Huisman e col. fizeram com êxito a separação cromatográfica de várias hemoglobinas, em carboximetilcelulose, conseguindo inclusivamente, com esta técnica, fraccionar a hemoglobina A nos seus componentes.

Electroforese. — À electroforese em coluna livre e depois em papel se deve o isolamento das hemoglobinas anormais que hoje conhecemos. Mais recentemente tem-se usado ainda a electroforese em gel, ou em pasta de amido, ou ainda em gelose.

No papel, as várias hemoglobinas, quando sujeitas a electroforese, dispõem-se de forma conhecida.

A observação dos diagramas electroforéticos de adultos normais mostrou que a hemoglobina A não é homogénea, contendo, além da fracção clássica, uma segunda fracção A_2, cuja mobilidade é sensìvelmente a da hemoglobina E, em tampão de veronal, pH 8,6. Nos indivíduos normais esta fracção não excede 3 % da quantidade total de hemoglobina, mas na talassemia ela pode subir a 15 % (isto quanto à *thalassaemia minor*, pois na *major* a fracção A_2 quase falta).

Mais recentemente verificou-se que, a par de casos de *thalassaemia minor* em que a concentração da fracção A_2 era normal, outros existiam nos quais o aumento desta fracção era a única anormalidade apresentada, não obstante deverem ser considerados heterozigotos para a talassemia, por serem pais de doentes de Cooley e, por fim, Silvestroni e Bianco observaram mesmo um doente em quem o diagnóstico clínico de doença de Cooley se impunha, em famílias onde o único estigma hemático era o aumento da fracção A_2.

Nas populações onde a talassemia e a hemoglobina E coexistem o aumento daquela fracção pode criar aparentes dificuldades de diagnóstico. Aksoy e col. salientam poder tomar-se como regra, em tais circunstâncias, que, quando a fracção que origina as dúvidas for inferior a 20 %, é provável que se trate de A_2 e não de E.

Para o diagnóstico diferencial das hemoglobinas que se comportam semelhantemente a PH 8,6 não devem perder-se de vista os seguintes factos:

As hemoglobinas S e D distinguem-se pela não transformação falciforme dos eritrócitos que não contenham a primeira e pela diferente mobilidade na electroforese em ágar, onde a hemoglobina D se separa da S, confundindo-se com a A.

As hemoglobinas L e Galveston (P) distinguem-se pela electroforese em coluna líquida e pela cromatografia em amberlite I R C 50.

A hemoglobina F distingue-se mal da A a pH 6,5 ou 8,6. Para melhor efectuar a sua separação pode recorrer-se a tampões mais alcalinos (pH 9 ou 9,2), mas a melhor forma de a separar de A é indubitàvelmente a cromatografia em amberlite I R C 50.

A hemoglobina M está ligada a uma anomalia da globina que implica defeituosa oxigenação do sangue. A mancha correspondente a esta hemoglobina é de cor castanho-achocolatada.

A hemoglobina Bart's, que a pH 8,6 se desloca como a hemoglobina N, a pH 6,5 fixa-se entre as hemoglobinas N e H e a pH 6 é a mais rápida de todas as variedades conhecidas.

Enfim, as hemoglobinas H e I têm idêntica mobilidade a pH 8,6, mas diferem a pH 6,5. Nestas condições, segundo Rucknagel, a hemoglobina H migra para o ânodo, enquanto a hemoglobina I se desloca para o cátodo.

DISTRIBUTION OF THALASSAEMIA AND OF ABNORMAL HAEMOGLOBINS

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There are now known, in addition to the two physiological haemoglobins, foetal haemoglobin (F) and normal adult haemoglobin (A), some 16 variants of adult haemoglobin and possibly two of foetal haemoglobin. A brief description of these variants will be given. Tropical medicine is particularly interested in those haemoglobins which give rise to disease. The most severe haemoglobinopaty is sickle-cell anaemia. Combinations of one gene for sickling with genes for other abnormal haemoglobins result in modified sickle-cell anaemia: sickle-cell haemoglobin C disease, sickle-cell haemoglobin D disease, sickle-cell haemoglobin E disease. Haemoglobin C, D and E diseases are much milder haemoglobinopathies.

Another type of haemoglobinopathy is one in which haemoglobin A is not replaced by another adult haemoglobin, but where its formation is inhibited as in the inherited condition thalassaemia. The homozygous state causes a severe anaemia, thalassaemia major, and the heterozygous state varies in severity from being completely innocuous to resembling thalassaemia major. Thalassaemia can occur in combination with abnormal haemoglobins and mixed syndromes are the result.

The geographical distribution of the abnormal haemoglobins shows them to be related to certain parts of the world and to certain races. Haemoglobin S is found particularly in Africa and in some distinct populations of the Near East and India. Haemoglobin C is localised in West Africa and haemoglobin D is found at an appreciable incidence only in India. Haemoglobin E is associated with

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the Siamese, Burmese, Malays and with their neighbouring populations. Haemoglobin H has been found more in Chinese than in any other group. The other haemoglobins are still «rare». As the haemoglobins are characters which play an active part in natural selection, it has to be assumed that their incidence will rise or fall according to whether they are of selective value under definite environmental stresses. The nature of some such stresses will be discussed.

TALASSEMIA

NUNO CORDEIRO FERREIRA
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É sobre a talassemia que iremos falar, deixando de lado a parte clínica da afecção, dado o interesse científico deste colóquio, que incide sòmente sobre o estudo dos problemas referentes às hemoglobinas.

Entre nós, desde que Cordeiro Ferreira publicou o primeiro caso, há vinte anos, foram já descritos bastantes casos de talassemia, sobretudo a forma *minor*.

Primeiramente afigura-se-nos com interesse saber se se pode considerar a talassemia como uma verdadeira hemoglobinopatia. Tem-se discutido muito sobre o assunto e os autores que a ele se têm dedicado ainda hoje não são da mesma opinião.

Como Trincão, pensamos que a não podemos considerar dentro de uma tal denominação, pelas razões que vamos expor:

Na verdade, na talassemia não existe uma hemoglobina anormal, mas apenas uma interferência na expressividade do gene responsável pela formação da hemoglobina A.

Em 1946 Vecchio descobriu que na doença de Cooley havia uma percentagem, em geral sempre elevada, de hemoglobina alcalinorresistente.

Choremis e col. verificaram que as concentrações dessa hemoglobina poderiam ser de 7 a 98 % da hemoglobina total, sendo, em média, superior a 50 %, mas que não havia relação entre a gravidade da doença e a concentração de hemoglobina alcalinorresistente,

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embora, em geral, os doentes com percentagens inferiores a 20 tivessem um quadro clínico menos grave.

Itano e col. confirmaram isto ao mostrarem, em duas famílias com membros afectados de doença de Cooley, que um caso grave tinha apenas 12 % de hemoglobina alcalinorresistente, enquanto noutro, leve, essa percentagem atingia o elevado número de 94 %.

Nas formas graves de talassemia existe sempre, por pequena que seja, uma certa quantidade de hemoglobina A, ao contrário do que se passa nos casos de hemozigotismo das hemoglobinopatias, em que há apenas hemoglobinas anormais e pequenas percentagens de hemoglobina F.

Por último, a maior parte dos autores não encontram diferenças entre a hemoglobina alcalinorresistente de Cooley e a hemoglobina fetal.

Penatti e col. verificaram a identidade destas hemoglobinas por meio dos métodos de cromatografia, mono e bidimensional, da absorção no ultravioleta e da desnaturação ácida pelo ácido acético N/4.

Chernoff demonstrou, por meio de reacções imunoquímicas, que estas duas espécies de hemoglobina alcalinorresistente são iguais.

Por outro lado, Roche e col., por meio da sua técnica de solubilidade das hemoglobinas, verificaram a identidade das mesmas.

Shapira, ao estudar o número e a natureza dos grupos N terminais da hemoglobina da anemia de Cooley, notou também a mesma igualdade nos grupos terminais da fetal.

Apenas Perosa e col. verificaram que havia diferenças entre estas duas hemoglobinas. Para estes autores há desigual velocidade de desnaturação pelos ácidos das correspondentes oxi-hemoglobinas. Também as respectivas carboxi-hemoglobinas têm velocidades de desnaturação diferentes, não só pelos ácidos, como também pelos alcalis. Verificaram também diferenças quando empregaram a electroforese em papel, em tampão de pH 9, e depois da passagem de uma corrente de 120 V, durante 12 horas, pois a hemoglobina alcalinorresistente de Cooley tem uma velocidade de migração compreendida entre a F e a A.

Apesar destes trabalhos de Perosa, a grande maioria dos autores afirmam que as hemoglobinas alcalinorresistentes fetal e da talassemia são idênticas.

QUADRO 1

1.º Na talassemia não existe uma hemoglobina anormal
2.º Não há relação entre a percentagem de hemoglobina alcalinorresistente e a gravidade da doença
3.º Existe mesmo nas formas graves hemoglobina A
4.º As hemoglobinas alcalinorresistentes da talassemia e fetal são idênticas

QUADRO 2 — *Comparação das hemoglobinas alcalinorresistentes da talassemia e fetal*

Autor	Método	Comportamento
Penatti e colab.	Cromatografia mono e bidimensional	Igual
Penatti e colab.	Absorção no U. V.	Igual
Penatti e colab.	Resistência aos ácidos (ácido acético N / 4)	Igual
Chernoff	Reacções imunoquímicas	Igual
Roche e colab.	Solubilidade	Igual
Shapira e colab.	Estudo dos grupos N terminais	Igual
Perosa e colab.	Resistência aos ácidos da oxi-hemoglobina	Diferente
Perosa e colab.	Resistência aos ácidos e alcalis de carboxi-hemoglobina	Diferente
Perosa e colab.	Electroforese em papel com tampão da veronal de sódio (pH 9) e passagem de corrente de 120 V durante 12 horas	Diferente

QUADRO 3 — *Hipóteses de Silvestroni e Bianco para explicarem as excepções à lei da transmissão hereditária da talassemia*

1.º Paternidade ilegítima
2 º Mutação
3.º Interferência de outras anemias hemolíticas
4.º Erros de diagnóstico

Em 1942 Gatto, depois de ter estudado 28 indivíduos pertencentes a famílias onde se tinham encontrado 8 doentes com anemia de Cooley, observou que se verificava sempre nos dois progenitores microcitose associada a aumento de resistência globular. Os doentes eram, pois, homozigotos à anomalia, enquanto os heterozigotos apresentavam a forma *minor* ou eram simples vectores assintomáticos da tara. Neel confirmou mais tarde os estudos daquele autor.

Silvestroni e Bianco, estudando 173 doentes de talassemia *major*, sòmente em 165 casais de progenitores encontraram o estigma talassémico. Como explicação para tais excepções à lei da transmissão da hereditariedade apresentam 4 hipóteses:

1 — Paternidade ilegítima.
2 — Mutação.
3 — Interferência de outras anemias hemolíticas.
4 — Erros de diagnóstico da anemia de Cooley em doentes com outras enfermidades, susceptíveis de com ela se confundirem clìnicamente.

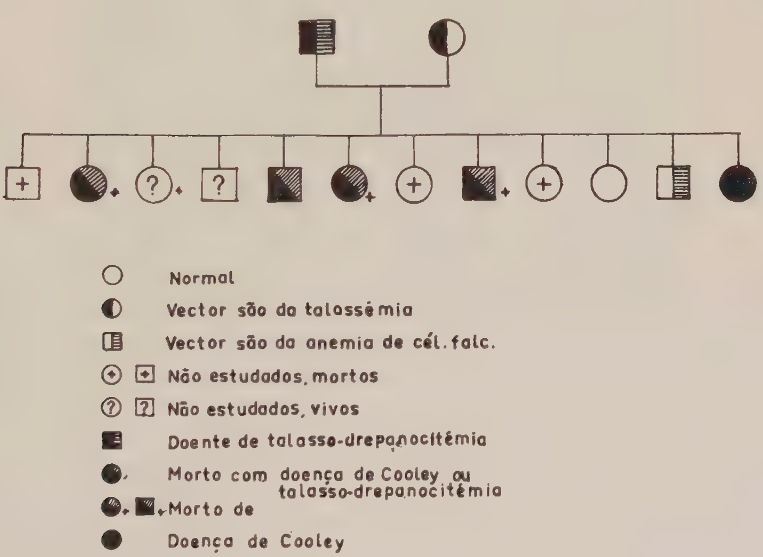
Normal
Vector são da talassémia
Vector são da anemia de cél. falc.
Não estudados, mortos
Não estudados, vivos
Doente de talasso-drepanocitémia
Morto com doença de Cooley ou talasso-drepanocitémia
Morto de
Doença de Cooley

Fig. 1

Está demonstrado que o gene talassemia não é alélico em relação ao da hemoglobina A e também ao das outras hemoglobinas anormais.

Basta para tal recordar as observações de Bianco e Silvestroni (fig. 1), em que existe um filho normal num casal em que o pai sofre de talassodrepanocitose e a mãe de estigma talassémico. Brown e col. (fig. 2) viram também que num casal, em que o pai apresenta uma combinação de talassemia-hemoglobinose C e a mãe é heterozigota para a drepanocitose, há um filho simultâneamente afectado pelas três afecções hemáticas.

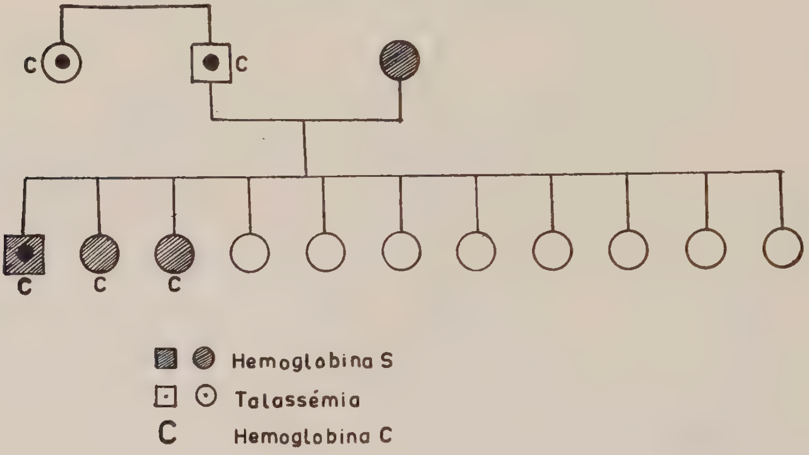

Fig. 2

Sabe-se que nos vectores heterozigotos da talassemia estão descritas todas as possíveis transições entre a forma grave da talassemia *minor* e as formas inaparentes. Gatto tenta explicar esse facto pela diferente expressividade do gene patológico.

De grande importância para o estudo da talassemia foi a introdução da electroforese em bloco de amido por Kunkel e col. Verificaram estes que, por meio desta técnica, decompunham a hemoglobina A em duas fracções, uma mais rápida e maior, outra mais lenta e menor, a fracção A2 (fig. 3).

Num estudo de 155 indivíduos normais verificaram estes autores que a percentagem desta fracção estava compreendida entre 1,8 e 3,2, com o valor médio de 2,54. Estudando, por último, a existência desta fracção em diversas hemopatias, observaram que sòmente nos portadores de tara talassémica esta mostrava alteração, estando quase sempre aumentada. Assim, em 34 portadores do estigma a média da fracção A2 foi de 5,11 %. Em doentes com a forma *major* a fracção A2 estava normal ou mesmo diminuída.

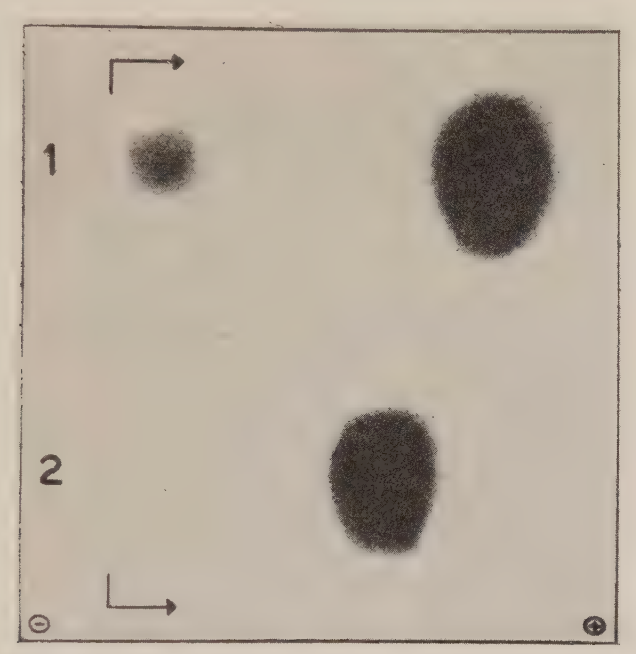

Fig. 3

Diamond notou que em 23 pais de doentes com anemia de Cooley a fracção A2 estava sempre aumentada, sendo os seus valores compreendidos (fig. 4) entre 3,3 e 6,8 %. Os valores (fig. 5) individuais eram constantes e sempre aproximados nos membros da mesma família.

Marioni, em 141 portadores de estigma talassémico, e Silvestroni, em 10 indivíduos afectados pelo que ele denomina *anemia microcítica constitucional,* obtiveram resultados semelhantes aos dos autores americanos. Silvestroni afirma que, mesmo em electroforese em papel, consegue separar, além da fracção A2, uma outra, A3, ainda mais lenta, e ainda uma outra mais rápida, que é mínima nos indivíduos normais, mas que aparece aumentada nos microcitémicos, sobretudo naqueles que foram esplenectomizados. Os valores da fracção A2 encontra-

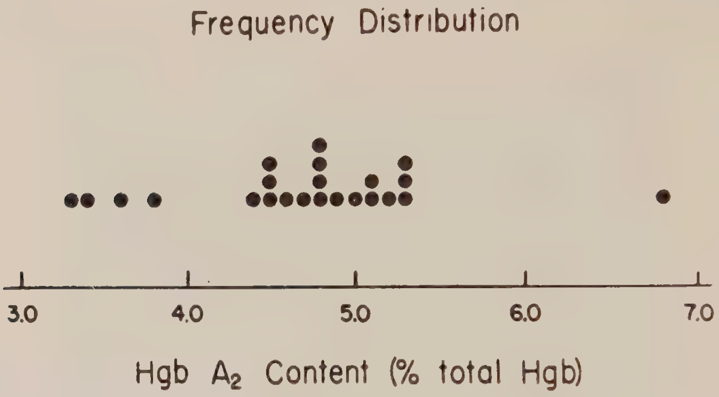

Fig. 4

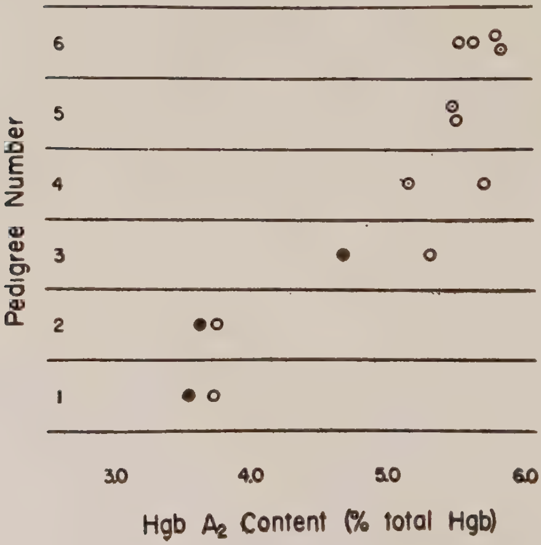

Fig. 5

dos por este autor, em 223 indivíduos normais, foram de 1,95 %, enquanto os valores médios achados nos microcitémicos foram de

4,22 %. A curva de frequência (fig. 6) das percentagens encontradas mostra que, quer nos normais quer nos microcitémicos, a distribuição se faz segundo uma curva de Gauss.

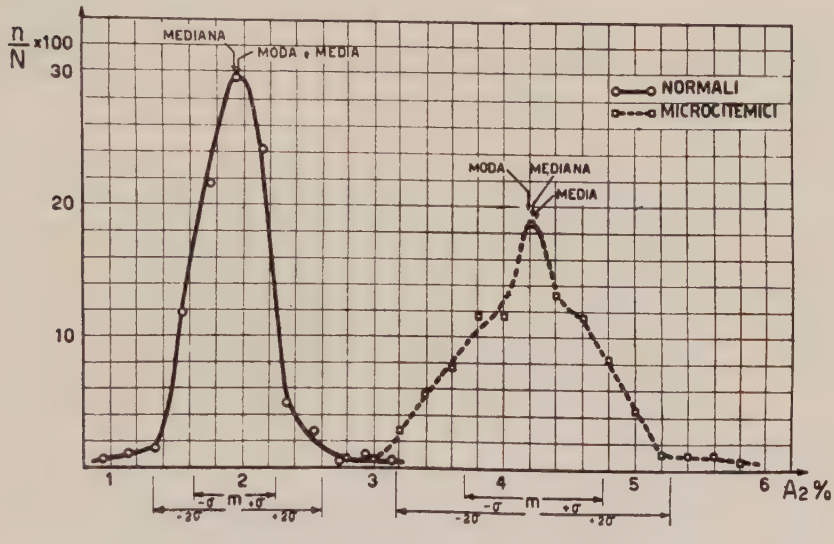

Fig. 6

Muito recentemente Huisman, por meio da cromatografia em carboximetilcelulose, observou também aumento da fracção A2 nos heterozigotos da talassemia.

QUADRO 4

Segundo Silvestroni:	
Volume corpuscular médio	98,84
Fragilidade osmótica	98,81
Contagem de glóbulos	91,90
Segundo Kunkel:	
Fracção A2 da hemoglobina	94,00

A existência quase constante de valores elevados de A2 nos portadores de estigma talassémico tem uma grande importância no diagnóstico desta afecção. Silvestroni afirma que os elementos mais seguros de diagnóstico dos portadores são, como podemos ver pelo quadro 4, em primeiro lugar, o volume corpuscular médio, diminuído em 98,84 % dos casos, em segundo lugar, a fragilidade osmótica, diminuída em 98,81 % dos casos, e, em terceiro lugar, a contagem dos glóbulos vermelhos, diminuída em 91,9 % dos casos. Ora Kunkel afirma que a fracção A2 está elevada em 94 % dos casos, portanto um valor da mesma ordem dos restantes.

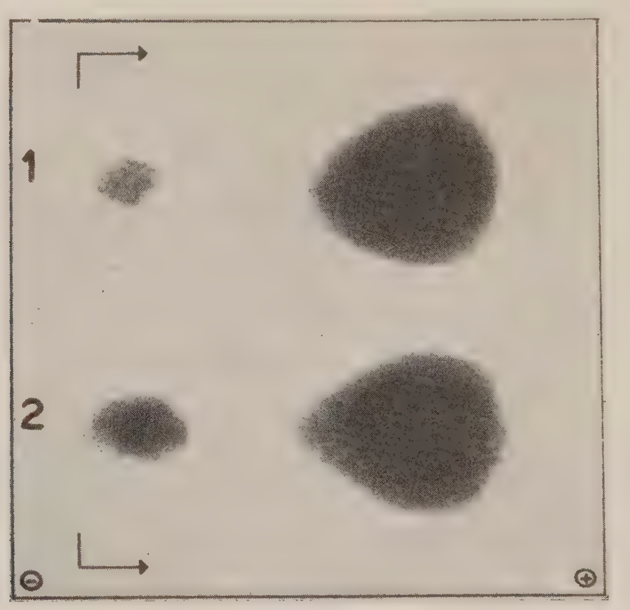

Fig. 7

Diz-nos Diamond que se pode fazer o diagnóstico de portador do estigma talassémico apenas pela determinação da fracção A2 e do volume celular médio. Descreve mesmo dois casos de microcitose em que foi excluída a hipótese de uma talassemia *minor* pelo não aumento daquela fracção. Para este autor é necessário, para o diagnóstico da talassemia *minor*, o aumento de A2, não sendo, porém, este elemento suficiente, pois Josephson descreve um caso de recaída de anemia perniciosa acompanhada de aumento desta fracção.

A explicação do aumento de A2 nos portadores do estigma talassémico é-nos dada por Kunkel, por analogia com os casos de associação de talassemia-hemoglobinose C ou S, em que a proporção das hemoglobinas A e anormal deixa de ser aproximadamente igual quando não existe o estigma, passando a haver um aumento da hemoglobina anormal quando aquele existe.

O gene talassémico interfere na síntese das hemoglobinas, e portanto na produção da hemoglobina A; daí um aumento da fracção A2. Quanto ao aparente paradoxo do não aumento da fracção A2 nos doentes de anemia de Cooley, explica-o o autor pela existência de hemoglobina fetal. Esta é produzida por um mecanismo independente do gene A, e, portanto, deve calcular-se a percentagem da fracção A2 apenas em relação à hemoglobina A existente no doente. Deste modo, a fracção A2 é sempre tão ou mesmo mais elevada nos indivíduos atacados pela doença do que nos portadores da tara.

Na talassemia, por meio da técnica de electroforese em bloco de amido, segundo Marione, podem observar-se os seguintes tipos de traçado:

1) Fig. 7 — Fracção A2 aumentada. Falta de hemoglobina F. Próprio dos portadores da tara.
2) Fig. 8 — Fracção A2 em valor absoluto diminuída. Hemoglobina F muito aumentada. Traçado próprio da maior parte dos doentes de Cooley.
3) Fig. 9 — Traçado onde pràticamente se encontra hemoglobina F. Próprio dos casos mais graves da doença de Cooley.

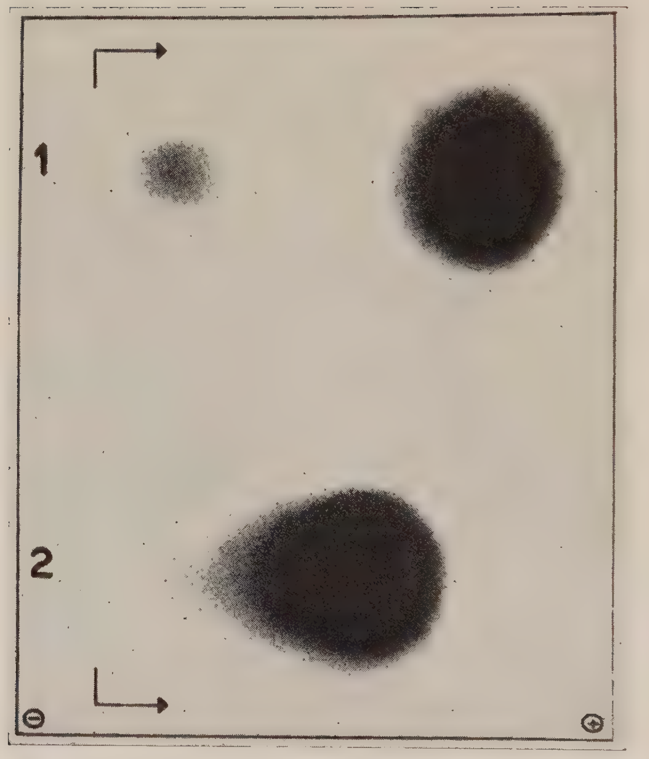

Fig. 8

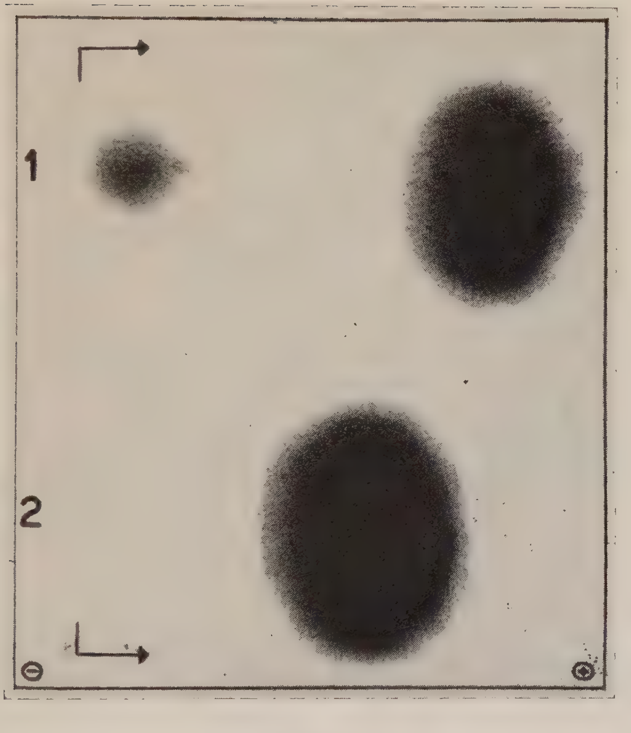

Fig. 9

LA PATHOLOGIE DE L'HÉMOGLOBINE C

RAYMOND J. CABANNES
Alger

Malgré sa grande diffusion dans les pays où la nigrescence est importante, l'hémoglobine C reste exclusivement attachée à la race nègre et plus particulièrement à certains individus originaires d'une partie bien définie de l'Afrique Noire, l'Afrique Occidentale et surtout le plateau Voltaïque.

Dans le premier cas d'homozygotisme, rapporté par Spaet, Alway et Ward (27), l'Hb C représentait la totalité du pigment héminique. Depuis, les cas similaires se sont multipliés et l'on connaît maintenant plus de 80 cas de porteurs d'Hb C maladie.

Le trait Hb C, expression hétérozygotique de l'hémoglobinose, intéresse un nombre de sujets beaucoup plus important que l'Hb C maladie. Dans certaines tribus de Haute-Volta nous avons mis en évidence jusqu'à 27 % d'Hb AC (23). Dans la totalité du plateau de la Volta, la moyenne des sujets porteurs d'Hb AC atteint 18 %. Cette proportion va en diminuant au fur et à mesure que l'on s'éloigne de ces régions. Cependant l'expansion de l'Hb C est limitée à la partie Ouest du continent africain. Au delà l'Hb C devient une curiosité, mis à part les territoires américains pour lesquels elle a pris, il y a bien longtemps, les bateaux des négriers.

Le trait Hb C est asymptomatique et sa découverte n'est possible que lorsqu'on se livre à des enquêtes systématiques ou lorsque l'on suspecte l'hémoglobinose chez un sujet atteint d'une maladie qui n'est pas due à l'hémoglobinopathie.

L'hémoglobinose C homozygote ou hémoglobine C maladie, elle-même, n'est pas incompatible avec la vie. Les sujets qui en sont atteints sont même voués à une existence de durée normale. Rarement quelques accidents peuvent appeler l'attention. On est donc relati-

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vement pauvre en documents anatomopathologiques ayant trait à cette maladie. Sur les 80 cas dont nous pouvons faire état, 25 seulement ont fait l'objet d'un examen clinique approfondi; parmi ces derniers, 6 ont été splénectomisés et un seulement autopsié. On a ainsi obtenu le matériel d'une étude anatomopathologique partielle.

Ce n'est qu'au travers de ces quelques cas qu'il a été possible de se rendre compte de la pathologie de l'hémoglobine C. Cet exposé repose donc sur des données fragmentaires et sur un petit nombre d'observations. Il ne saurait rapporter toutes les modifications pathologiques dues à l'Hb C. Il représente seulement un jalon dans l'étude qui ne manquera pas d'être complétée au fur et à mesure de la révélation de nouveaux porteurs homozygotes d'hémoglobine C.

90 % des sujets porteurs d'Hb C maladie sont des adultes. Quelques enfants ont pu être examinés, mais ils étaient déjà à une période assez avancée de leur vie (10 mois, 5, 9 et 11 ans). Nous échappent donc tous les enfants morts en bas âge et tous les morts-nés issus de parents porteurs de la tare.

Les signes ante-mortem se confondent avec les symptômes cliniques de constatation courante dans cette affection. L'Hb C maladie ne conduisant pas fatalement à la mort, il est impossible de préciser les signes précurseurs d'une échéance malheureuse. Aussi allons-nous rapporter les signes cliniques les plus évidents, tels que nous pouvons les mettre en lumière à la fois par l'examen clinique du patient et par toute la gamme d'examens paracliniques: radiographie, électroencéphalographie, hématologie et anatomopathologie.

Chez l'enfant, les manifestations pathologiques dues à l'Hb C sont le plus souvent rares ou peu importantes.

Spaet, qui a le premier décrit l'Hb C maladie chez un enfant de 5 ans, a noté des signes cliniques frustes (27).

Singer a observé deux enfants de 9 et 11 ans qui ne présentaient aucune manifestation morbide (26).

Nous avons, nous-mêmes, décelé seulement une anémie d'intensité moyenne chez le nourrisson homozygote que nous avons examiné avec Portier (17).

Chez le nouveau-né et le nourrisson, les désordres causés par l'Hb C sont inexistants. Motulsky (29), qui a vu apparaître progressivement la fraction C au cours des jours ou des semaines suivant la naissance, apporte ainsi l'explication de cette carence de signes cliniques; l'hémoglobinose ne se trouvant en fait réalisée qu'au moment où tout le stock d'hémoglobine fœtale a été remplacé par du pigment adulte.

Cependant, certains de nos enfants présentant une hémoglobinose AC, sans stigmates de thalassémie, montrent une anémie importante. Celle-ci, apparente, occupe toute la scène. On ne sait si elle est l'expression précoce de l'hémoglobinopathie ou un phénomène surajouté. Nos petits malades présentent alors des crises hémolytiques latentes. Par ailleurs, cette anémie se superpose le plus souvent à un état de carence nutritionnele fruste, courant dans nos pays. L'hémoglobinose interviendrait alors pour fragiliser le système sanguin de l'enfant. A moins que l'état de carence ne potentialise les phénomènes hémolytiques, provoquant une anémie érythroblastique sévère qui pourrait être fatale.

C'est chez l'adolescent ou chez l'adulte qu'apparaissent les manifestations les plus sévères et les plus révélatrices de l'hémoglobinose C. Ce sont surtout des troubles anémiques: pâleur, dyspnée, fatique, hors de proportion avec le chiffre globulaire, qui n'est pas très bas. Se manifestent également des douleurs de l'hypochondre gauche.

L'examen d'un patient chez lequel on a révélé de l'Hb C en totalité permet la constatation d'un certain nombre de signes que l'on retrouve chez tous les porteurs de cette hémoglobinopathie:

L'*anémie* est toujours d'intensité moyenne marquée surtout par la pâleur des téguments et des muqueuses;

La *splénomégalie* est de volume variable, mais elle est constante. Depuis la grosse rate atteignant la crête iliaque gauche jusqu'à la rate débordant légèrement le rebord costal, tous les intermédiaires existent. Cette rate est ferme, régulière, légèrement douloureuse spontanément et à la palpation.

Le foie est généralement de volume normal, mais il peut être légèrement hypertrophié. Il n'existe pas de signes biologiques d'insuffisance hépatique.

Les appareils cardiovasculaire et rénal sont normaux.

Les crises douloureuses abdominales sont quelquefois expliquées par la présence d'une lithiase vésiculaire (2 cas).

Les crises douloureuses articulaires ne sont accompagnées d'aucun symptôme inflammatoire local.

Le système osseux est sensiblement normal. Edington & Lehmann (4) ont signalé une «décalcification des os du crâne». Il semble que cette décalcification soit plus diffuse et qu'elle atteigne notamment les épiphyses des os longs. Elle explique, en partie, le retentissement de l'Hb C sur la croissance et le développement général des sujets. Nous verrons plus loin que l'image de décalcification s'interprète anatomiquement par une hyperplasie médullaire.

Il paraît plus important de souligner la participation du système nerveux. Les premières relations sur ce sujet ont été faites par H. Ranney (19, 20) (cas de paralysie cérébrale de type choréo-athétosique) et par J. L. Morgan (débilité mentale). Reprenant les travaux de Sarrouy et Portier sur les «syndromes neuro-hémolytiques» (24), nous avons avec Portier et ses collaborateurs pratiqué des examens électroencéphalographiques à des sujets porteurs d'Hb C. Nous avons ainsi mis en évidence des modifications de l'E.E.G. latentes qui se manifestent par:

«Sur les tracés de repos: tendance à la labilité corticale caractérisée par des rythmes de fréquences variables, instables, survenant par bouffées irrégulières. Il existe également des éléments de type irritatif localisés ou diffus sous forme de pointes ou de pointes-ondes.

À l'épreuve à l'hyperpnée: on voit apparaître rapidement des ondes lentes, monorythmiques de grande amplitude, généralisées. Ces bouffées d'ondes lentes persistent longtemps (jusqu'à 2 et 3 minutes) après l'arrêt de l'épreuve» (18).

Ces altérations sont surtout marquées chez les sujets agés. Cependant elles sont présentes chez les sujets jeunes et chez les enfants et les nourrissons, qu'ils soient homozygotes ou hétérozygotes.

Enfin, ces altérations du tracé E.E.G. diffèrent totalement de celles qui ont été enregistrées par Portier et ses collaborateurs dans la thalassémie, ainsi que de celles décrites par Hill, Hugues & Camp-Davies dans la drépanocytose.

Les maladies intercurrentes ont une action importante sur l'expression pathologique de l'hémoglobinose C. En effet, ces afections, surtout si elles sont de nature inflammatoire, ont un retentissement révélateur qui se manifeste par une aggravation de l'anémie avec érythroblastose périphérique atteignant jusqu'à 15 et 20 % des leucocytes circulants. C'est au cours d'une pleurésie séro-fibrineuse, d'apparence banale, que nous avons mis en évidence notre première observation d'Hb AC (17).

Nous ne possédons actuellement aucune observation de femme porteur d'Hb CC en état de grossesse. Nous ne connaissons donc pas encore l'influence de la gestation sur l'hémoglobinose C maladie. De même, nous ne savons rien de l'influence de l'Hb C sur la grossesse. Chez la femme enceinte porteuse d'Hb AC les observations que nous avons relevées ne signalent aucune anomalie.

La splénectomie d'une part et la ponction de moelle osseuse d'autre part ont fournit les premières données anatomopathologiques concernant l'hémoglobinose C.

Si la ponction osseuse pour l'examen de la moelle est d'une pratique facile et courante, la splénectomie n'a pu être faite que dans certaines conditions. Aussi les cas d'ablation de la rate sont-ils peu nombreux. Nous en connaissons 6 dont les examens histologiques ont permis d'intéressantes constatations.

Les rates ainsi prélevées étaient toujours hypertrophiées, mais de volume et de poids variables:

Pour Diggs & coll. (3)	2,3 kg
Pour Hartz & Schwartz (5)	0,72 kg
Pour Singer & coll. (26)	0,8 kg
Pour Wheby & coll. (30)	1,0 kg
Pour Portier, coll. & nous-mêmes (18)	2,1 kg et 1,2 kg

Jensen & coll. ont trouvé à l'autopsie de leur malade décédé une rate pesant 0,575 kg (9).

Ces rates étaient toutes adhérentes, entourées de périsplénite. La pulpe rouge a toujours présenté le même aspect, malgré les auteurs: elle apparaît comme gorgée de sang, très congestionnée, de teinte rouge foncé.

Du point de vue histologique:

L'architecture de la rate est conservée, l'image trabéculaire est normale.

La pulpe rouge montre une congestion intense des cordons de Billroth (Diggs, Singer, Wheby, Portier). Les artères peuvent être normales (Portier, Singer, Hartz), présenter un endothélium suspect (Diggs) ou une hypertrophie avec artériosclérose modérée et hyalinisation discrète (Wheby).

La pulpe blanche est normale ou peu abondante, avec appauvrissement des centres lymphatiques, marqué par une décoloration de ces centres clairs (Singer, Diggs). Quelquefois on a pu noter la présence d'une hémorragie au centre d'un follicule ébauchant un nodule de Gandy-Gamma (Portier).

Au total:

Congestion considérable de la pulpe rouge avec engorgement des sinus par des érythrocytes et quelquefois présence de petits agrégats globulaires semblant former une masse homogène (Diggs).

Appauvrissement de la pulpe blanche.

Ces rates n'évoluent pas vers la sclérose. Leur charge en pigments ou hémosidérine est peu importante. L'érythrophagocytose est rare.

La moelle osseuse présente le même aspect dans l'hémoglobinose C homozygote ou hétérozygote. On y trouve chaque fois une hyperplasie importante où prédominent les éléments érythroblastiques. Cette érythroblastose est toujours de type normoblastique. Elle est strictement médullaire avant la splénectomie. Celle-ci déclenche toujours une érythroblastose périphérique importante, pouvant aller jusqu'à 30 % des éléments blancs circulants.

Il a fallu la première et seule autopsie pratiquée par Jensen & coll. (9), chez un sujet atteint d'hémoglobinose C maladie, pour vérifier anatomiquement ce que la clinique et les explorations fonctionnelles laissaient supposer.

Pour la rate et la moellle osseuse on retrouve évidemment les mêmes modifications que nous avons signalées plus haut.

Le foie est le siège d'une congestion portale peu importante par des érythrocytes. Dans le parenchyme hépatique et les cellules de Kuppfer on met en évidence du pigment brun qui donne les réactions biochimiques du fer. Il s'agit d'hémosidérine dont la concentration à cet endroit traduit un processus hémolytique de degré relativement modéré.

Tous les gros vaisseaux des poumons renferment des caillots. Certains vaisseaux plus petits contiennent des caillots déjà anciens, recanalisés, renfermant des dépôts importants d'hémosidérine. D'autres vaisseaux encore sont obstrués par des caillots plus récemment constitués. Ceux-ci sont en voie de prolifération fibroblastique. On assiste donc à un processus thrombosique progressif atteignant successivement plusieurs vaisseaux pulmonaires. Cependant le parenchyme semble intact. Il ne présente pas, en particulier, de zones infarcies.

Les reins montrent une fibrose sévère. L'intima des artérioles rénales se hyalinise progressivement et même prolifère d'une manière paradoxale.

Enfin le cerveau présente des aires de dégénérescence corticale avec coloration jaunâtre de la partie latérale et antérieure de la corne antérieure du ventricule.

En résumé:

Congestion splénique;
Hyperplasie érythroïde de la moelle osseuse;
Thrombose progressive des vaisseaux pulmonaires;
Fibrose et hyalinisation rénales;
Dégénérescence corticale du cerveau;
Hémosidérose localisée au foie et aux caillots en voie de résorption ou de réorganisation.

DISCUSSION

Dans l'hémoglobinose C l'hémolyse est peu considérable et contraste encore avec la discrétion des signes biologiques. Il semble qu'à cause de son importance minime elle soit facilement résorbée, permettant l'élimination des produits d'hémolyse sans fixation par les tissus ni hypersidérose. Cette hémolyse ne se produit d'une manière importante qu'à des endroits très localisés, au moment où se forment des caillots qui évolueront vers la sidérofibrose. Les produits d'hémolyse sont alors retenus localement ou versés dans la circulation. Ils ne sont pas très abondants à cause de l'échelonnement dans le temps des phénomènes thrombosiques, ainsi que le montrent les examens anatomopathologiques. Malgré l'engorgement sinusal dont elle est le siège, la rate elle-même fixe peu les produits d'hémolyse. Ceux-ci sont sans doute retenus par les éléments du foie, puis éliminés par la bile, ainsi qu'en témoignent les chiffres élevés de stercobiline fécale trouvés par les auteurs. Cette élimination n'est toutefois possible que parce que les phénomènes hémolytiques sont peu importants. Cependant ceux-ci peuvent, malgré leur allure latente, se manifester quelquefois avec plus de vigueur; c'est le cas pendant les maladies inflammatoires ou infectieuses ou ; l'occasion d'un épisode carentiel chez les enfants. Il semble qu'à ce moment la fragilité des hématies soit exagérée.

L'érythroblastose normoblastique qui accompagne ces phases de déglobulisation mériterait un commentaire. Aucun argument autre que l'hémolyse ne peut expliquer ce phénomène malgré les caractères paradoxaux que nous avons déjà signalés avec Portier (18). Après la splénectomie l'érythroblastose périphérique semble être le résultat de la levée de l'inhibition splénique sur la diabase médullaire.

Jusqu'à ce jour aucune explication de cette hémolyse n'a été avancée. On n'a pas davantage essayé de fixer le mécanisme des lésions constatées. L'hémolyse est, à notre avis, secondaire à l'engorgement du système vasculaire par des érythrocytes porteurs d'une hémoglobine anormale, l'Hb C, qui leur confère un comportement anormal. Ce comportement est le résultat des anomalies suivantes:

1) Anomalie de la forme des érythrocytes: ceux-ci se présentent, dans une grande proportion, sous forme de cellules cibles ou «target cells».
2) Présence de cristaux intraérythrocytaires mis en évidence spontanément dans l'Hb CC et provoqués en athmosphère anoxique dans l'Hb AC.
3) Diminution et étalement de la résistance globulaire osmotique.
4) Diminution de la durée de vie des érythrocytes chargés d'Hb C transfusés à un sujet normal — les hématies normales ayant une durée de vie normale chez un sujet porteur d'Hb C.

Ainsi les globules rouges chargés d'Hb C sont morphologiquement modifiés; ils sont fragiles; ils ont une durée de vie écourtée;

ils sont apparemment dépourvus de l'élasticité physiologique normale des globules rouges. Ils se comportent donc comme des masses inertes facilement agglomérables. La stase ou l'engorgement vasculaires découlent de cette propriété. L'hémolyse, comme Ponder (15) l'a souligné, est le résultat de cette stase qui a sa preuve dans la congestion splénique, le processus thrombosique pulmonaire et l'ischémie cérébrale engendrant la dégénérescence corticale.

Les cristaux intracellulaires, produits en anoxie, sont-ils la cause de cette inertie des globules rouges et de leur agglutinabilité passive? Ils ne sont pas spécifiques de l'Hb C. Il semble en effet, d'après Lehmann, que toutes les Hb anormales — sauf Hb S — soient susceptibles de produire des cristaux intraérythrocytaires dans certaines conditions d'anoxie. Cependant, en l'état actuel de nos connaissances, il faut tout de même admettre que la cristallisation peut, comme la falciformation, être un obstacle à la libre circulation des hématies et favoriser les agglomérats intravasculaires.

Ainsi les modifications pathologiques constatées dans l'hémoglobinose C sont liées à trois ordres de faits interdépendants: biochimique, physique et anatomique.

L'Hb C constitue-t-elle un caractère anthropologique devenant pathologique hors des conditions climatiques réalisées pour les nègres de Haute-Volta par exemple? Il est troublant de constater que l'Hb C n'a pas encore frappé par ses manifestations cliniques les médecins de ces pays comme l'a déjà fait la sicklémie. Il faut alors penser que les globules chargés d'Hb C se comportent normalement dans un organisme acclimaté. Car, enfin, les seuls cas d'Hb CC ayant conduit à la découverte de l'hémoglobinopathie ont été détectés hors des territoires pouvant être considérés comme «le nid» de l'Hb C. Nous avions été frappés par l'analogie électrophorétique existant entre l'Hb du dromadaire de nos pays et l'Hb C. Un certain temps nous avions cru que cette ressemblance pouvait être le fait d'une identité moléculaire. Nous avons ensuite émis l'hypothèse que cette Hb aurait pu être modifiée par mutation génétique sous l'influence des conditions de vie.

Tout ceci n'est qu'hypothèse et demande à être verifié. Dans ce domaine tout reste à faire.

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HEMOGLOBINAS ANORMAIS NAS PROVÍNCIAS PORTUGUESAS DE ÁFRICA

A. R. NOGUEIRA

Os primeiros inquéritos relativos à pesquisa de hemoglobinas anormais nos territórios ultramarinos portugueses limitaram-se à pesquisa de hemoglobina S por qualquer das provas pelas quais se provoca transformação falciforme dos eritrócitos.

As prospecções deste tipo, feitas entre 1944 e 1958, permitiram a observação do sangue de 30 119 indígenas das cinco províncias portuguesas de África.

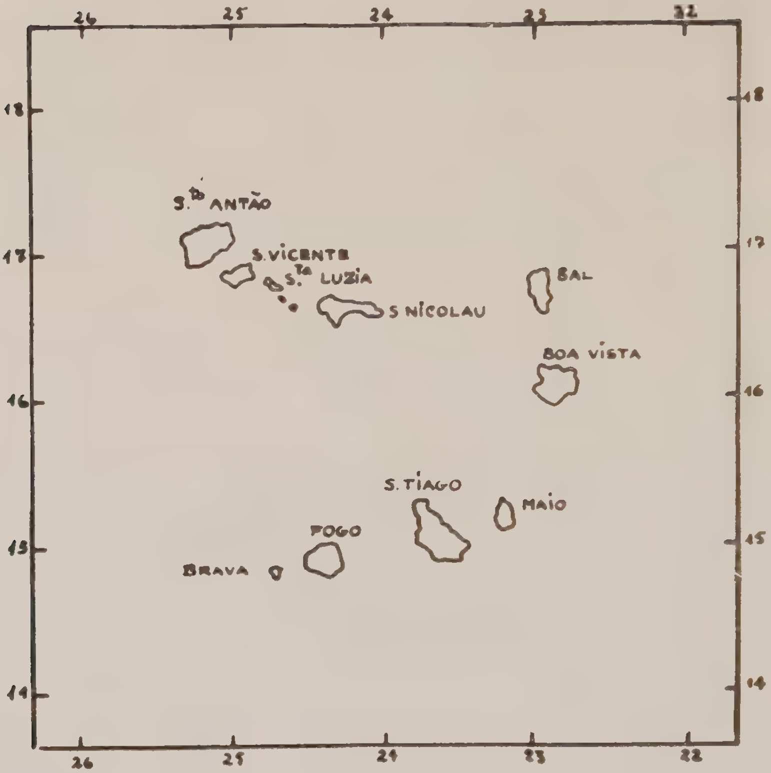
Desde 1956 que começaram a estudar-se hemoglobinas de nativos do ultramar português pela electroforese em papel, à qual se associou a pesquisa da transformação falciforme pela técnica de Emmel.

Vamos dar conta das investigações feitas, ao mesmo tempo que pomos em confronto com os nossos dados os resultados obtidos na simples pesquisa de falciformação.

CABO VERDE

O quadro 1 mostra os resultados da pesquisa directa de drepanocitose por Pinto Nogueira e col. e os obtidos por Trincão e col. num grupo de cabo-verdianos que em 1956 trabalhavam na ilha do Príncipe e ali foram observados. A percentagem de hemoglobina S é maior na população de Santiago, que tem características negróides mais acentuadas.

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 403-411, 1959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BRAVA

Fig. 1

QUADRO 1 — *Drepanocitose em Cabo Verde*

Pesquisa directa

Autores	Ilhas	Observações	Positivos
Pinto Nogueira e col.	S. Antão	2063	3,39 %
	Brava	437	1,37 %

Pesquisa directa e electroforese da hemoglobina

Autores	Ilhas	Observações	Positivos
Trincão e col.	Todas	897	5,2 %
	Santiago	453	7,2 %
	Brava	198	6,0 %

Não se encontraram outras hemoglobinas anormais.

GUINÉ

Damos em primeiro lugar os resultados da pesquisa directa de drepanocitose nesta província por vários observadores e em seguida os que obtivemos por electroforese em papel da hemoglobina de 9224 indivíduos das principais tribos.

Em ambos os inquéritos são as tribos do litoral aquelas em que a percentagem de drepanocitose é menor. Pelo contrário, nas tribos do interior esta percentagem é bastante mais elevada.

QUADRO 2 — *Drepanocitose na Guiné*

Pesquisa directa

Autores	Tribos	Observações	Positivos
Fraga de Azevedo e col.	Futa-fula	31	16,0 %
Trincão e col. (1950)	Papel	500	3,0 %
Salazar Leite e col.	Manjaco	500	3,2 %
	Balanta	500	5,0 %
	Felupe	466	1,7 %
	Baiote	473	1,3 %
	Nalu	501	2,7 %
	Saracolé	286	8,3 %
	Mandinga	500	15,0 %
	Beafada	505	15,2 %
	Pajadinca	358	18,4 %
	Fula-forro	500	23,0 %
	Fula-preto	430	22,0 %
	Futa-fula	515	17,4 %

Observaram-se ainda alguns indivíduos provenientes de tribos nómadas que vivem principalmente em territórios estrangeiros e cujo número não permite conclusões sobre a verdadeira incidência de hemoglobinas anormais entre os seus componentes.

Além da hemoglobina S, encontraram-se as hemoglobinas C, K e N.

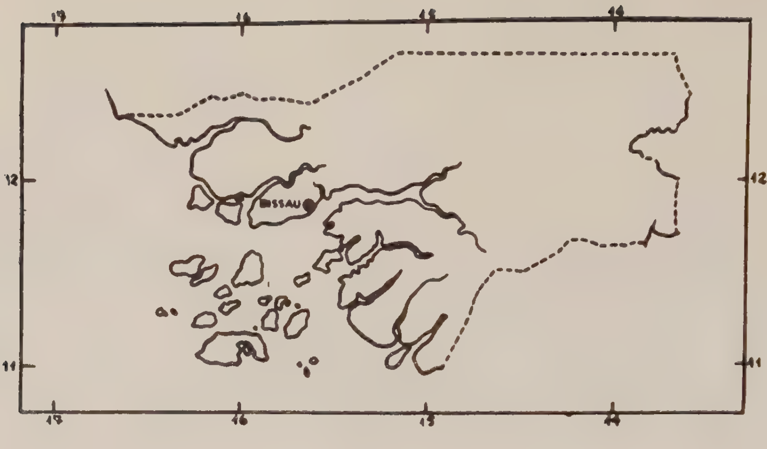

Fig. 2

QUADRO 3 — *Hemoglobinas — Guiné, 1958*

Raças	Número de casos					Percentagens			
	Total	AS	AC	AK	AN	AS	AC	AK	AN
Papéis	1244	16				1,28			
Fulas	1553	244	6	2	4	15,71	0,38	0,12	0,26
Balantas	776	7		1	1	0,90		0,12	0,12
Bijagós	632	3		1	1	0,47		0,15	0,15
Mancanhas	534	1			1	0,19		0,25	0,19
Mandingas	895	104	10			11,6	1,11	0,11	
Manjacos	758	15			3	1,97		0,13	0,43
Nalus	100	6				6			
Sossos	94	3			2	3,1			2,1
Saracolés	268	44	3			16,4	1,11		
Beafadas	797	102	1	1	1	12,7	0,12	0,12	0,12
Pajadincas	300	48		1	1	16		0,33	0,33
Cassangas	144	34				23,6			
Suruás	10	2							
Landomãs	3								
Nar	2								
Felupes	641	36				5,6			
Baiotes	342	1				0,21			
Banhuns	63	9				14,2			
Tilboncas	8	3	1						
Mieforas	1								
Timenés	4								
Jacancas	43	4							
Laibes	3								
Bagas	8								
Jalofos	1								
Total	9224								

S. TOMÉ E PRÍNCIPE

Dos indivíduos deste grupo, uma parte dos naturais de S. Tomé foram observados na vizinha ilha do Príncipe, onde se encontravam contratados para trabalhos agrícolas. Outros foram estudados no decurso duma visita à ilha de S. Tomé.

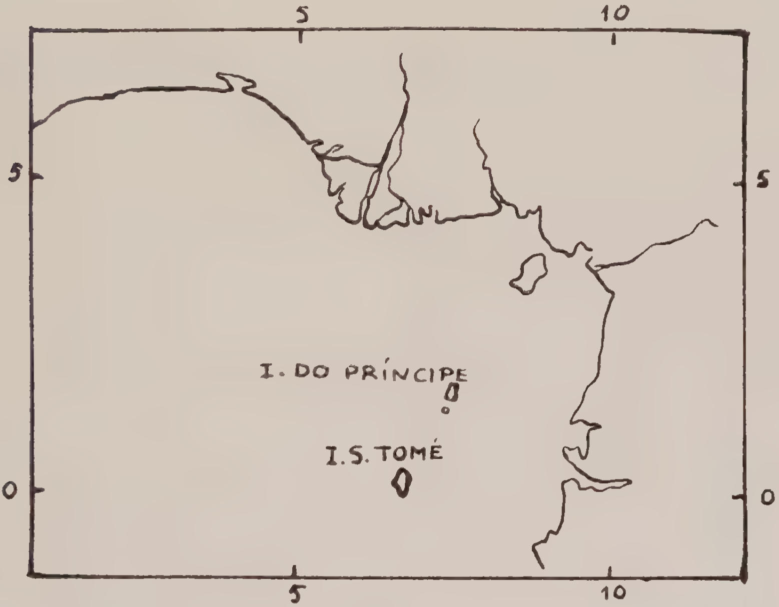

Fig. 3

QUADRO 4 — *Hemoglobinas anormais em S. Tomé e Príncipe*

Autores	Ilhas	Observações	Hb S (%)	Hb C (%)	Hb K (%)
Trincão e col.	S. Tomé	422	14,2	3	0,47
	Príncipe	380	22,1	3	—

Apesar da proximidade, a população do Príncipe está de há muito fixada na ilha e, pràticamente, não se cruza com a da ilha vizinha.

Talvez por este motivo, a percentagem de hemoglobina S é ali nìtidamente mais elevada. Encontraram-se ali também as hemoglobinas C e K.

ANGOLA

Também aqui damos separadamente os resultados das observações directas doutros investigadores. As nossas pesquisas por electroforeses em papel, num núcleo de soldados angolanos prestando ser-

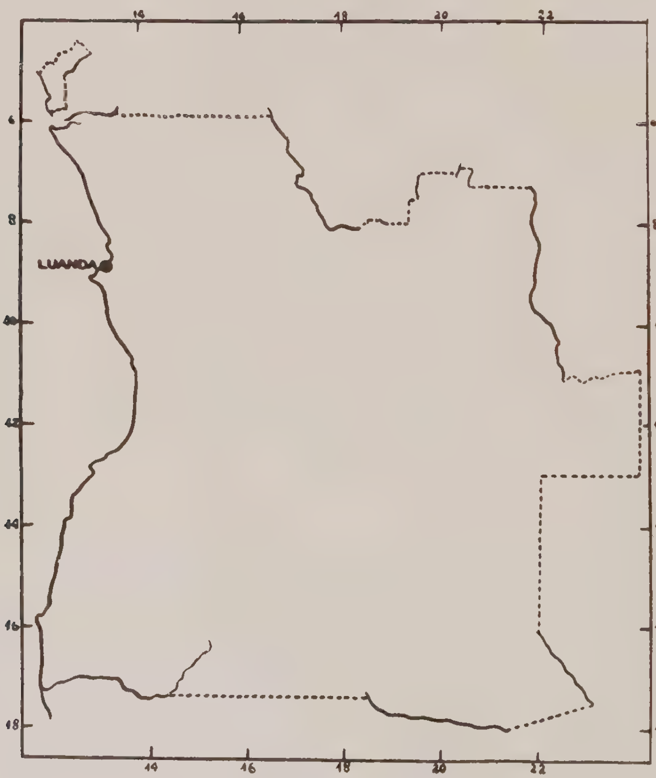

Fig. 4

QUADRO 5 — *Drepanocitose em Angola*

Pesquisa directa

Autores	Tribos	Observações	Positivos
Salazar Leite e col.	—	1973	7,29 %
Sarmento	—	216	8,3 %
Valdemar Teixeira	—	186	27,85 %
Trincão e Almeida	—	36	8,8 %
Trincão e Almeida	Bacuandos	43	4,6 %
	Guissis	70	10,0 %
	Curocas	27	3,7 %
	Mútuas	41	4,8 %
Salazar Leite e Ré	Bosquímanos	249	0,0 %
Santos David	Lunda	605	19,17 %
	Quioco	1709	16,97 %
	Songo	542	24,72 %
	Minungo	280	20,71 %
	Xingo	290	21,38 %
	Bangala	98	43,88 %
	Luena	88	11,36 %
	Luimbo	72	18,06 %
	Caconga	135	24,44 %
	Matala	61	27,87 %
	Outras	53	15,09 %
Moura Pires	Quioco	1000	21.3 %

viço na ilha do Príncipe, deu uma percentagem de 12,3 % de heterozigotos SA, não se tendo encontrado outras hemoglobinas anormais.

MOÇAMBIQUE

Damos os resultados das investigações directas de Foy e col., em comparação com as que fizemos por electroforese em papel em trabalhadores moçambicanos observados na ilha do Príncipe.

As percentagens de hemoglobina S que nestas condições se encontraram diferem sensìvelmente das de Foy e col.

Mesmo admitindo que os dados relativos a indígenas do Centro e Sul da província não sejam significativos por ser pequeno o número de indivíduos observados, a divergência em relação aos indígenas naturais de regiões ao norte do Zambeze é muito nítida.

Os nossos casos provieram principalmente da zona costeira do Norte daquele rio e a maioria dos indígenas afirmavam pertencer à *tribo dos Macuas*, justamente onde Foy e col. encontraram uma percentagem de falciformação de 38 %!

Não devem os nossos resultados ser afectados por endogamia, visto os trabalhadores moçambicanos contratados para prestar serviço na ilha do Príncipe se oferecerem voluntàriamente para esse trabalho e, portanto, provirem das mais variadas regiões, não sendo aparentados entre si.

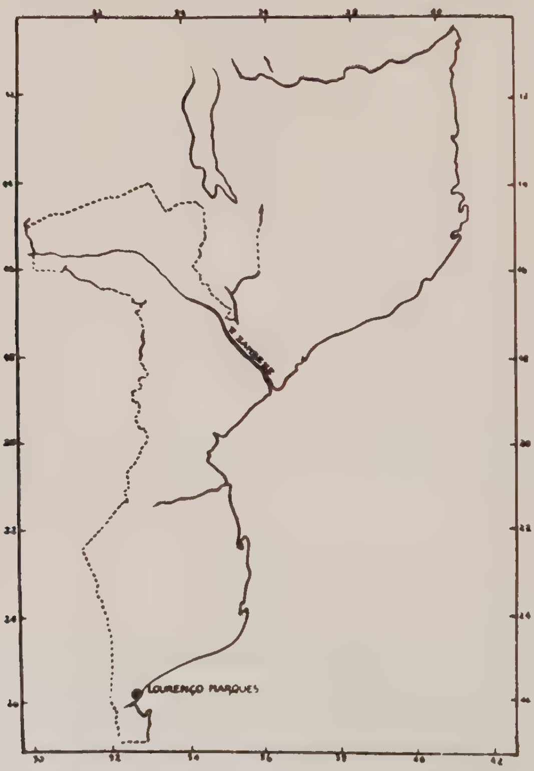

Fig. 5

QUADRO 6 — *Drepanócitos em Moçambique*

Pesquisa directa

Autores	Regiões	Tribos	Observações	Positivos (%)
Foy e col.	Norte	Macondes	100	40
	»	Macuas	111	38
	»	Lomués	100	4
	»	Chuabos	100	3
	»	Eratis	25	0
	»	Manhanas	50	4
	»	Maravias	25	0
	Norte e Centro	Senas	50	1
	Norte e Sul	Tachanas	50	2
	Centro	Angonis	50	0
	»	Machanganas	150	2
	Centro e Sul	Vátuas	100	2
	Sul	Muchopes	100	2
	»	Mungones	50	1
	»	Bongas	100	1
	»	Tongas	100	1
Lessa e Dessai	»	Landins	186	0,53

Pesquisa directa e electroforese

Autores	Regiões	Observações	Positivos %
Trincão e col.	Norte	389	3,5
	Centro e Sul	61	9,8

Não se encontraram outras hemoglobinas anormais.

Dado que as percentagens de falciformação apontadas por Foy e col. são das mais altas do mundo, apenas explicáveis se a tribo a que dizem respeito vivesse isolada dos povos circunvizinhos, o que se não verifica, julgamos antes que a pseudofalciformação tenha viciado aqueles resultados.

THE DISTRIBUTION OF SOME RARE HEMOGLOBINOPATHIES IN LIBERIA

FRANK B. LIVINGSTONE [1], WOLF W. ZUELZER [2], ABNER R. ROBINSON [2]
and
JAMES V. NEEL [3]

During the past two years my associates and I have conducted a hemoglobin survey of the native tribes of Liberia. In this period paper electrophoresis has been done on some 2,000 bloods, fetal hemoglobin determinations on about 1,500, and moving boundary electrophoresis, solubility determinations, and other tests, on fewer numbers.

Hemoglobin S (sickle-cell hemoglobin) was found in approximately 7 % of all subjects, but the frequency varied considerably among the different tribes. In Northwestern Liberia the tribes have 15-20 % hemoglobin S carriers; in Central Liberia, the frequency is about 12 %; and in Southeastern Liberia, it is only 1 %. Solubilitiy tests were done on most of the bloods which showed an AS pattern on paper electrophoresis and only a single one had the solubility of hemoglobin D. Thus, hemoglobin D seems to be somewhat rarer in Liberia than in the American Negro, among whom Chernoff found 4 hemoglobin D carriers out of 1,000. In contrast to other parts of West Africa, hemoglobin C was also found to be quite rare in Liberia; only 8 persons with hemoglobin C being found in the entire survey. In addition to these widespread hemoglobins our survey encountered three rare hemoglobinopathies which I would now like to discuss in greater detail.

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The first two of these are abnormal hemoglobins which migrate faster than normal hemoglobin in an alkaline buffer. When first discovered, they were called Liberian 1 and Liberian 2. Since then several attempts have been made to determine their relationship to other fast-moving hemoglobins which have already been assigned letters, but their exact status si still in doubt. Liberian 1 is similar to hemoglobin J, but at present does not appear to be identical to it, while Liberian 2 does appear to be identical to hemoglobin K, which has been found by Cabannes in Algeria and by Lehmann and Edington in Ghana.

Liberian 1 was found 8 times in the 2,000 samples for an overall frequency of 0.4 %. However, in the Mande tribes of the interior of Central Liberia it had a frequency of about 2 %; 2 of 159 for the Loma, 3 of 214 for the Kpelle, and 2 of 155 for the Mano and Gio combined. A single other example was found in the Gola tribe for a frequency of 1 %. Since the Gola border on some of the Mande tribes which have higher frequencies, this finding could very easily be due to mixture. Thus, it appears that Liberian 1 is restricted to several tribes in Central Liberia and is not found in the tribes in the north and south of the country. In addition, Allison did not find it in Sierra Leone nor has our group found it in the Ivory Coast to the East of Liberia.

Liberian 2 was only encountered twice in our survey and both these persons came from tribes in Central Liberia; one was Bassa and the other, Kpelle. The subjects were questioned on the tribes of their immediate ancestors and no evidence for tribal mixture could be obtained for either the parental or grandparental generation.

The question then arises as to how many mutations are responsible for these occurrences of Liberian 1 and Liberian 2. Because of the rarity with which these abnormal hemoglobins are found throughout the world, it would appear that the mutation rates are very low. This is more evident when one considers that the heterozygote for either of these genes and the normal allele does not have any pronounced clinical symptoms, and thus appears to have normal fitness. In fact the reproduction histories of these individuals seems to indicate that they are more fit than normals. Thus, there does not appear to be much selection against these genes, and, if the mutation rates were appreciable, one would expect to encounter them more frequently. Since the mutation rates do appear to be very low, it would seem most reasonable to assume that all the examples of each abnormal hemoglobin found in Liberia are due to the same mutation. The distribution of Liberian 1 in several tribes at about the same frequency seems to indicate that this hemoglobin is an ancient inheritance of the peoples in this part of Liberia. Since it is found in tribes with quite different ethnic backgrounds, it has undoubtedly taken many generations to become distributed as it is today. This also seems to imply that the Liberian 1 gene does not have a very great selective advantage associated with it, since, if it did, one would expect it to have attained a higher frequency in this time. Thus, the value of about 2 % which is found among these diverse tribes would appear to be the highest the equilibrium frequency could be. In this way this gene, and most likely the gene for Liberian 2, are not like the genes for hemoglobins S, C, and E, which are found in very high frequencies in some parts of the world.

The third hemoglobinopathy which our survey discovered seems at present to be different from the two previously discussed. Among the Kru tribes of Eastern Liberia, we have found several adults who have increased amounts of fetal hemoglobin, which in some cases is as high as 45 %. Clinically these individuals do not seem to have symptoms associated with this condition, although, as is usual in Liberia, their hemoglobin values are below what is considered normal in Europe or the United States. In four tribes of Eastern and Central Liberia the frequency of adults having more than 10 % fetal hemoglobin was about 1 %; 1 of 125 for the Kru, 1 of 69 for the Grebo, 1 of 72 for the Krahn, and 1 of 100 for the Mano. These tribes surround the Webbo tribe, in which the frequency was found to be 5 of 140 or 4 %; hence this distribution seems to indicate the spread of this characteristic from the Webbo tribe. Although I have used the term *tribe* for the Webbo peoples, they are the most isolated peoples in Liberia and actually have no overall tribal organization or even tribal consciousness. They are divided into several small groups of towns which are under one chief. In one such chiefdom, the frequency of adults with greater than 10 % fetal hemoglobin was found to be 3 of 15 or 20 %. Although this sample is very small, it does seem to indicate that this characteristic can attain high frequencies, and thus might have some selective advantage associated with it. It thus appears to be different from Liberian 1 and Liberian 2, and like hemoglobins S, C, and E in this respect.

THE CLINICAL DIAGNOSIS OF SICKLE-CELL ANAEMIA IN CHILDREN

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INTRODUCTION

Sickle-cell anaemia in West Africa is to a great extent a disease of children. Most doctors, when they first come to the area, find it difficult to distinguish between true sickle-cell anaemia and anaemia from other causes associated with the sickle-cell trait. This distinction can only be made with absolute certainty by special haematological techniques, particularly paper electrophoresis of haemoglobin, but these are not available everywhere and it is the object of this paper to show that the diagnosis can be made with a fair degree of certainty in most cases from clinical considerations alone.

The paper is based upon the study of 96 patients with sickle-cell anaemia who have attended the Children's Department of University College Hospital, Ibadan, where a special clinic is run for them. These patients have now been under observation for periods of up to five years and in all cases the diagnosis has been confirmed by paper electrophoresis.

AGE INCIDENCE

This is shown in a slide which was prepared from the records of 73 patients in whom an estimate could be made of the age of the patient at the time of the first onset of symptoms. It shows

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that symptoms most commonly develop during the second half of the first year and the second year of life.

SEX INCIDENCE

This is generally regarded as equal. In the present series 52 (54.2 %) of the patients were males and 44 (45.8 %) females.

SYMPTOMS

Pain in the limbs is the most characteristic feature of sickle-cell anaemia. It occurs in irregular attacks and varies in severity from a mild ache to pain of great severity. It is felt more frequently in the lower limbs than the upper, is felt in the bones rather than in the joints and is often confined to the hands or feet. Doctors who are unfamiliar with sickle-cell anaemia usually diagnose either acute rheumatism or leukaemia when they see such a patient for the first time. Another cause of pain in the limbs in these children is acute osteitis, which is a very common complication of both sickle-cell anaemia and sickle-cell haemoglobin C disease. Indeed the association is so common in Nigeria that it is now our practice to do sickling tests upon the blood of all children with acute osteitis.

Abdominal pain is another common symptom. Usually it is not very severe, «dragging» in character and attributable to the weight of the enlarged liver and spleen. Two of the patients developed acute abdominal symptoms for which laparatomy was performed, but in neither patient was any significant pathological condition found.

Abdominal enlargement is more common than pain. In many cases it is due to the massive enlargement of the liver and spleen which frequently occurs, but it is also partly due to the exaggerated lumbar lordosis producing undue prominence of the abdomen.

Jaundice is a common complaint but *pallor* is rarely mentioned since it is not obvious on superficial examination of the African child.

It is surprising how few symptoms can be attributed to the anaemia itself. Children with haemoglobin levels of about 5.0 g per 100 ml are often found to be attending school normally and their parents notice no more than a little lassitude.

SICKLE-CELL CRISIS

Children with sickle-cell anaemia are liable to sudden attacks of acute symptoms which may be associated with a fall in the haemoglobin level. The commonest symptoms of a crisis are fever and severe pain in the limbs and it has been noticed that a period of anorexia may precede its onset. Occasionally a crisis may take the form of a sudden fall in haemoglobin with few other symptoms.

Of our 96 patients, 51 are known to have had one or more such crises and 45 have had none.

A few of the patients presented for the first time in an anaemic crisis, but this is unusual and it is commoner for them to attend first on account of the less dramatic symptoms of the disease.

PHYSICAL SIGNS

General appearance. — The appearance of these patients is sometimes so characteristic that it may be possible to establish the diagnosis at a glance although this is exceptional. They are often tall and thin with an exaggerated lumbar lordosis and prominent abdomen. Their arms and legs are long and thin and their fingers long and tapering. The most characteristic feature is the shape of the head, which often shows bossing in the parieto-occipital regions giving it a square, flat-topped appearance. Less commonly there is frontal bossing as well producing a «hot-cross bun» appearance.

Pallor. — Some degree of mucosal pallor is usual since these children, when in average health, generally have a haemoglobin of 6 g to 8 g per 100 ml. In severe cases the mucous membranes appear blanched.

Jaundice. — A slight degree of scleral jaundice was found in 36.5 % of the patients and severe jaundice is not infrequent. A few of them show severe, persistent jaundice associated with biochemical evidence of advanced liver damage. Liver biopsy upon two such patients showed that they had already developed hepatic fibrosis, but after several years observation both are surprisingly well.

Cardiac abnormalities. — Abnormal cardiac signs were met with in 35 % of the patients, the incidence increasing with age. They were generally of a minor character, such as a slight degree of cardiac enlargement with the apex beat between the mid-clavicular and anterior axillary lines, or an apical systolic murmur. In only two of the patients was there a greater degree of enlargement. Heart failure was only met with as a complication of severe anaemia.

Enlargement of the liver and spleen. — These were not always palpable and the degree of enlargement varied from «just palpable» to enormous organs reaching to below the umbilicus. Both were usually firm and not tender.

Limbs. — Swelling or tenderness of the arms and legs is common. It may be slight and transient or associated with infection of the bone. This complication may be no more than a periostitis which settles down after a week or so, but severe osteitis occurs not infrequently, being about 80 times as common in sicklers as in non-sicklers. (Osteitis in sickle-cell anaemia is recognised as being ± 80 times as common as in non-sicklers.) A frequent and characteristic finding is swelling of the limbs not unlike that seen in juvenile rheumatoid arthritis. Similar changes may be seen in the toes, but a commoner finding is tender swelling of the dorsum of the foot.

HAEMATOLOGICAL FINDINGS

Simple haematology can be a considerable help in establishing the diagnosis of sickle-cell anaemia. The important findings are:

1. The demonstration of filamentous sickling.
2. The presence of nucleated red cells, target cells and sickle-cell forms in films of the peripheral blood.
3. A raised leucocyte count.

RADIOLOGICAL FINDINGS

These are very diverse and only a few of the more conspicuous ones can be mentioned:

1. *Skull.* — This commonly shows increase in the diplöeic markings due to marrow hyperplasia. Radial spiculation is occasionally seen but is not as constant a feature of sickle-cell anaemia as of thalassaemia.
2. *Long bones.* — The changes are best seen in the hands and feet where the metacarpals, metatarsals and phalanges show thinning of the cortex and trabeculae due to marrow hyperplasia and small translucent areas due to infarction. Periostitis is often seen and changes due to bone infection may be present.

SUMMARY

SALIENT POINTS IN THE ESTABLISHMENT OF A CLINICAL DIAGNOSIS

1. *History.* — A history of «rheumatic» pains in a West African child should be regarded as due to sickle-cell anaemia until proved otherwise.

2. *Physical examination:*

   a) Anaemia.
   b) Limb swellings and tenderness, especially swelling of the hands and feet in young children.
   c) Physique, posture and shape of head.

3. *Laboratory findings:*

   a) Demonstration of filamentous sickling.
   b) Presence of nucleated red cells, target cells and sickle-cell forms in films of the peripheral blood.

4. *Progress.* — Persistent anaemia with no response to antimalarials or heamalinus in a child whose blood shows sickling.

L'HÉMOGLOBINE ALCALINO-RÉSISTANTE AU MOYEN-CONGO: SON INTÉRÊT DANS L'ANÉMIE DRÉPANOCYTAIRE

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En dehors du sang du fœtus, ou du nourrisson dans les premiers mois de sa vie, on trouve une fraction d'hémoglobine douée de résistance à la dénaturation par les alcalins (Hb RDA):

1° Chez l'adulte normal, à un taux faible (2 %) et fixe.
2° Dans les hémoglobinoses héréditaires, soit dans:

- *a*) La thalassémie (Vecchio, 1948), à des taux moyens de 30 à 70 % dans la forme majeure;
- *b*) L'anémie drépanocytaire, de condition homozygote (Singer et coll., 1951);
- *c*) D'autres homozygotismes: EE, DD, CC, à des taux plus faibles et d'une manière inconstante;
- *d*) Des doubles hétérozygotismes: SC, ou, surtout, thalasso-drépanocytose.

3° Dans la sphérocytose héréditaire, parfois.
4° Parfois aussi, dans des hémopathies acquises; son excès est toutefois rarement signalé et toujours à des taux peu élevés.

On ne trouve pas d'Hb RDA dans les hétérozygotismes, par ex. AS.

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[1] The Author did not attend the meeting.

Dans la sicklanémie, c'est Singer et ses collaborateurs qui ont montré, en 1951, que cette fraction pouvait se trouver en excès. Parmi les travaux français, citons les études biochimiques de Roche, Derrien et leurs collaborateurs.

D'importants travaux ont aussi étudié cette Hb RDA dans la thalaso-drépanocytose.

À l'heure actuelle, il est admis dans la plupart des publications que le sang des sicklanémiques contient souvent une certaine quantité d'Hb RDA, pouvant représenter 20 % de l'Hb totale. C'est, par exemple, se qu'admet Lehman (1957).

Le but de cette communication est de rechercher la présence de l'Hb RDA chez des enfants souffrant d'anémie drépanocytaire (taux, variations éventuelles) et de confronter sa présence ou son absence avec les examens cliniques, biologiques et les enquêtes familiales. Nous l'avons aussi recherchée chez d'autres anémiques et dans divers échantillons de sang de la populacion de Brazzaville.

MOYENS D'ÉTUDE — RÉSULTATS

Pour chaque échantillon d'Hb, nous avons fait une électrophorèse sur papier, un dosage de fraction RDA (technique de Roche et Derrien) et, lorsqu'il existait de l'Hb S, une mesure de la solubilité par le test d'Itano. Chez presque tous les sicklanémiques, l'Hb des parents a été étudiée.

Nos résultats sont les suivants:

1° *Dans la sicklanémie.* — Sur 35 enfants, âgés de 1 à 14 ans, le taux de l'Hb RDA était 17 fois normal (de 0 à 1,5 %) et 18 fois excessif, allant de 6 à 24 % (15 fois entre 6 et 12 %). Les dosages ont été répétés, une ou plusieurs fois, chez 15 enfants, à des intervalles de 6 semaines à 6 mois:

   a) Chez 7 enfants elle était toujours présente; on a noté des variations, mais peu importantes (de 6 à 10 %, par ex.); elles paraissent souvent être provoquées par des crises hémolytiques, le taux de RDA s'élevant lors de la réglobulisation;

b) 5 malades n'ont jamais eu d'excès de RDA, même au cours, ou après, des crises hémolytiques (l'un d'eux a été examiné 5 fois en 6 mois, l'autre 4 fois en 5 mois);

c) 3 autres ont varié d'un taux normal à 5, 7 et 10 % (2 fois au cours de la réparation d'une crise hémolytique).

En somme, l'Hb RDA est en excès chez plus de la moitié des sicklanémiques; d'autres paraissent avoir un taux toujours normal.

2° *Dans le trait drépanocytaire.* — Sur 120 hétérozygotes, le taux a toujours été normal. Insistons sur le fait que nous n'avons pas rencontré de type SAF.

3° Sur 20 anémiques, dont 15 enfants ankylostomés, la RDA était normale. Un enfant anémique avec élliptocytose constitutionnelle n'en avait pas.

4° Enfin 150 dosages systématiques chez des enfants et adultes non porteurs du trait S ont montré un taux normal.

Sur plus de 300 recherches, nous n'avons donc trouvé d'Hb RDA que chez certains sicklanémiques.

L'HB RDA DANS LA SICKLANÉMIE — CONFRONTATIONS AVEC LES DONNÉES CLINIQUES ET BIOLOGIQUES.

D'une manière très générale, chez les malades ayant une fraction RDA en excès, on note:

Une légère atténuation de l'anémie et des crises douloureuses;

Une hépatomégalie et une splénomégalie plus fréquentes (les ponctions biopsies du foie ne nous ont jamais montré de foyers d'érythropoïèse extramédullaire);

Une augmentation habituelle de la solubilité de l'Hb au test d'Itano. L'examen des hématies par le test d'Emmel montre une falciformation plus lente. Dans les solutions hypotoniques, la résistance globulaire est, peut-être, légèrement augmentée (en ce qui concerne l'hémolyse terminale);

Enfin, les altérations radiologiques osseuses sont un peu plus importantes.

Tout ceci n'a rien d'absolu, et comporte des exceptions: formes bénignes ss RDA, ou splénomégalies sans RDA, par exemple.

Par ailleurs, nous n'avons pas trouvé de relations avec le pourcentage de sidéroblastes dans la moelle, ni avec la valeur globulaire (ce qui aurait pu suggérer un trouble de l'utilisation du fer, comme dans la thalassémie).

Les enquêtes n'ont pas montré de différences parmi les parents de sicklanémiques avec ou sans RDA.

COMMENTAIRES

Ainsi, en pratique, nous n'avons rencontré d'Hb RDA que chez les sicklanémiques, certains paraissant en avoir toujours, et d'autres jamais. On ne peut donc considérer la présence de cette fraction comme un phénomène banal au cours de la réparation d'une anémie. Peut-être s'agit-il d'une Hb de vicariance.

Le premier point consiste à rechercher l'intrication d'un gène thalassémique chez les malades avec excès de RDA. Ceci nous paraît très improbable:

Absence de signes cliniques ou hématologiques évoquant la microdrépanocytose;

Parents de ces malades de type AS; pas d'excès de RDA dans la famille;

Résultats de 1.050 électrophorèses systématiques, qui ont montré environ 20 % d'AS. Nous aurions trouvé quelques faux homozygotes si la thalasso-drépanocytose existait à Brazzaville;

Absence d'excès de RDA lors des recherches systématiques;

Absence de formes majeures ou mineures de la thalassémie dans notre région.

Le deuxième point serait de savoir si cette fraction RDA des sicklanémiques est une Hb de type fœtal, ou non. Les uns admettent en effet qu'il s'agit de la persistance, ou de la reprise, de l'hémoglobinogénèse fœtale. Pour Roche et Derrien, l'Hb RDA des drépanocytaires appartiendrait, au moins en partie, à la petite fraction de l'Hb adulte qui présente la propriété de l'alcalino-résistance, et qui existe à un taux faible et stable chez l'adulte normal.

Hb fœtale ou non, il reste à préciser les raisons de sa présence chez des sicklanémiques. A la suite de Rich, beaucoup pensent, comme Singer, que l'hémoglobinogénèse fœtale est inhibée, dans les conditions normales, par le gène A. Le gène S, à dose double, n'exercerait qu'une inhibition incomplète, d'où présence d'une certaine quantité d'Hb RDA. Cette théorie s'accorde de la présence de cette fraction dans le double hétérozygotisme SC et de son absence dans l'hétérozygotisme AS.

Quoiqu'il en soit, il nous semble que chez certains siclanémiques cette élaboration d'Hb RDA ne se produise jamais. Ceci nous conduirait à admettre, de ce point de vue, l'existence de deux sortes de maladie drépanocytaire:

Dans l'une, l'Hb A est entièrement remplacée par l'Hb S; il n'y a pas d'Hb RDA.

Dans l'autre, l'Hb S représente 75 à 95 % du total, le reste étant constitué par une fraction alcalino-résistante, et qui serait inégalement répartie dans les GR pour Singer et Fischer. Le rapport Hb S/Hb RDA varie chez le même malade, peut-être sous l'effet des crises hémolytiques. Chez ces sujets, la fraction RDA pourrait alors être interprétée comme une Hb de vicariance (pour Besnard et coll., chez le lapin rendu anémique, la synthèse de l'Hb RDA serait plus rapide).

En somme, un peu plus de la moitié de nos homozygotes SS paraît avoir une aptitude congénitale à synthétiser 2 sortes d'Hb, en dehors de tout gène thalassémique. On pourrait soulever l'hypothèse de l'intervention d'un facteur génétique, différent de celui de l'anémie méditerranéenne (maladie inexistante au Moyen-Congo), qui pourrait exercer son effet chez certains sicklanémiques, c'est-à-dire en l'absence du gène A.

En conclusion: nous avons trouvé chez plus de la moitié de nos sicklanémiques la présence d'une certaine quantité d'Hb alcalino-résistante. Seules des analyses approfondies de l'Hb et des enquêtes génétiques résoudront le problème posé par la présence de cette fraction.

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2ND SESSION: *Saturday, September, 6th — 9:00 to 10:30 a.m. Room A*

The meeting opened under the chairmanship of Dr. B. G. Maegraith.

The Rapporteurs were Dr. A. W. Woodruff and Dr. Carlos P. Trincão and the Secretary was Dr. Egídio R. R. Gouveia.

The following papers were presented and discussed.

SOME TECHNICAL ASPECTS ON THE DETERMINATION OF HAEMOGLOBINOPATHIAS

J. H. P. JONXIS

Now that the practical importance of the occurrence of abnormal forms of haemoglobin in the human blood has become evident the interest in them is no longer limited to a small group of specialists. The carrying out of the determination of abnormal haemoglobins has taken its place in many laboratories of clinical chemistry. Therefore it may be worthwhile to give here a short survey of the different methods which are available.

Although the determination of abnormal haemoglobins or of a too high amount of foetal haemoglobin is of primary importance, a proper diagnosis can only be made when also the most important haematological data are available. In many cases it may be necessary to study the family as well as the patient; if possible, we shall try to get data about 3 generations.

I will limit myself to a discussion of the techniques for the determination of the normal and the abnormal haemoglobins. We shall have to select our methods depending on the haemoglobin we are expecting to encounter. When we like to study, for example, the heterogeneity of the haemoglobin of a normal adult, we shall choose starch-electrophoresis, carboxymethylcellulose or Amberlite I.R.C.-50 chromatography; for the study of abnormal haemoglobins, paper-electrophoresis; for that of fœtal haemoglobin, alkali denaturation;

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for the differentiation of Hbs, S and D solubility. If possible, we shall use two different techniques so that their results might be compared.

The most important techniqes available at this moment are:

Alkali denaturation. — This method makes a quantitative determination of fœtal haemoglobin in a Hb-mixture possible. There are two forms: the precipitation techniques in which the denaturated haemoglobin is precipitated (1, 2), and the method in which the amount of undenaturated haemoglobin is spectrophotometrically determined during the denaturation-process. Since the denaturation of fœtal haemoglobin proceeds as a monomolecular reaction the velocity of the denaturation-process and the concentration of fœtal haemoglobin in the sample before the start of the process can be calculated (3, 4) (Figs. 1 and 2).

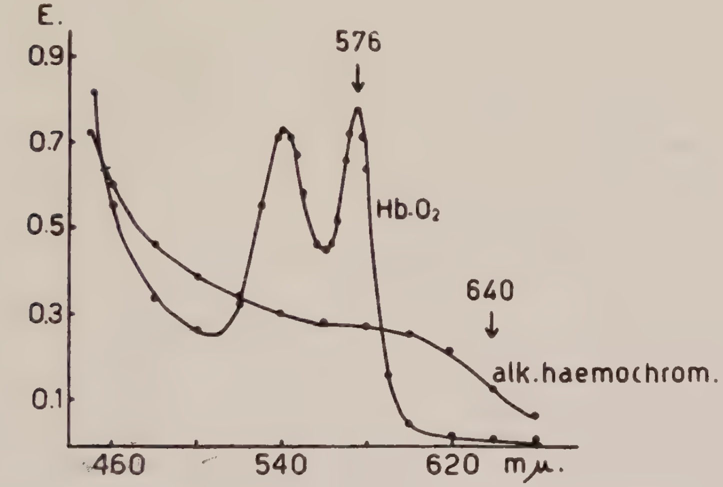


Fig. 1

The precipitation method of Singer has the advantage of being simple and taking less time. The accuracy, especially for high and low values of fœtal haemoglobin (5), however, is not great (Fig. 3).

The optical methods give somewhat more accurate values, especially for low fœtal haemoglobin concentrations, but even here concentrations lower than 2 % of fœtal haemoglobin cannot be measured with accuracy. For the detection of low concentrations of fœtal haemoglobin the immunological technique of Chernoff may be used (6).

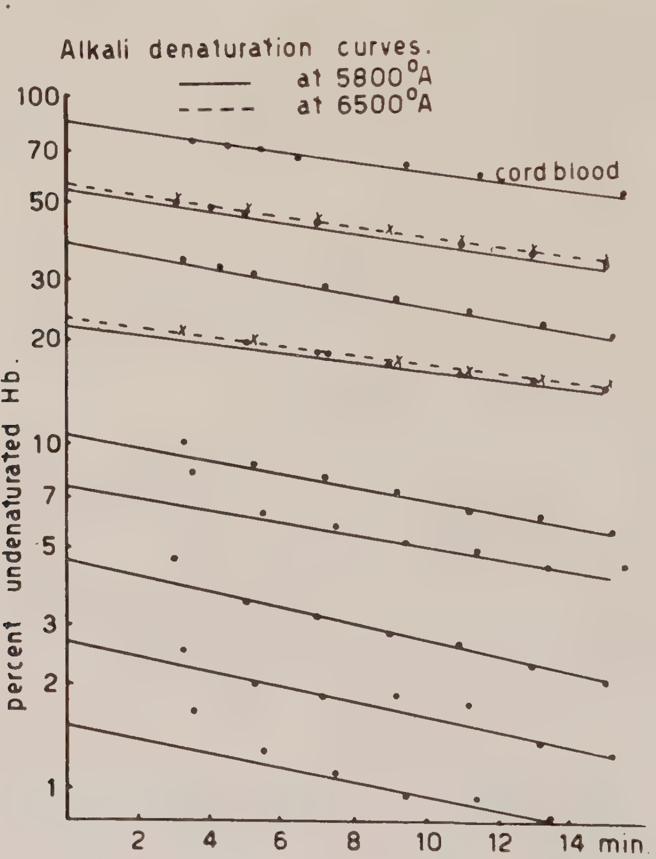


Fig. 2 — Denaturation curves of blood with different percentages of fœtal Hb. *Horizontal axis:* time in minutes. *Vertical axis:* percentage of undenaturated Hb

An abnormal haemoglobin with a resistance against alkali between those of fœtal and adult haemoglobin has been found recently by Lehmann (7). This makes it especially important to study the velocity with which the alkali denaturation of a haemoglobin sample takes place.

The methods for the determination of abnormal haemoglobins are in the first place the electrophoretical methods.

Tiselius moving boundary electrophoresis may give important information, but the procedure is difficult and time consuming; therefore most laboratories are using:

Paper electrophoresis. — For this method different forms of apparatuses are available which give good results in the hands of skillful workers (8).

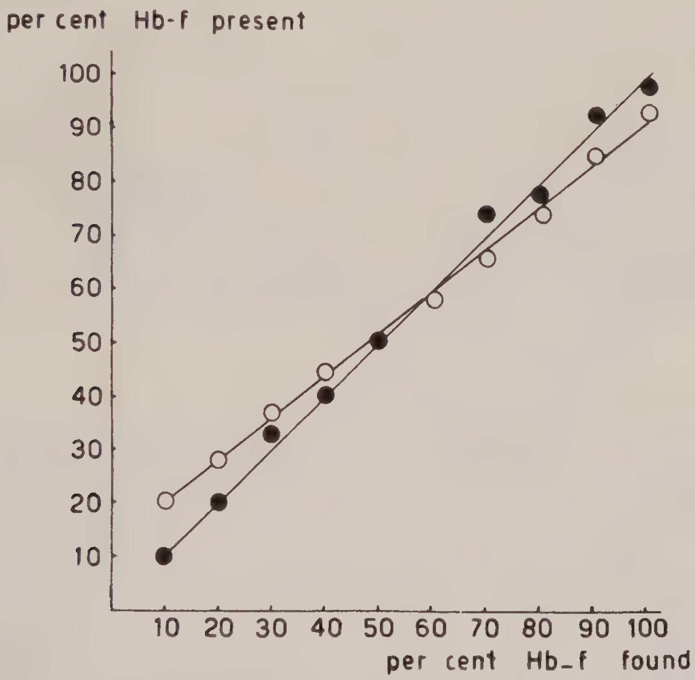


Fig. 3 — The results of the determination of fœtal Hb. *White spots:* one minute precipitation method. *Black spots:* optical method

We can distinguish the apparatuses into:

a) Apparatuses in which the paper strip is clammed between glass plates in order to prevent evaporation (Fig. 4);

b) Apparatuses in which the space around the paper strips is saturated with water vapour. In most of such apparatuses the strip is hanging down in the buffer solution (hanging strip method) (Fig. 5), in others the paper is held taut in a horizontal position.

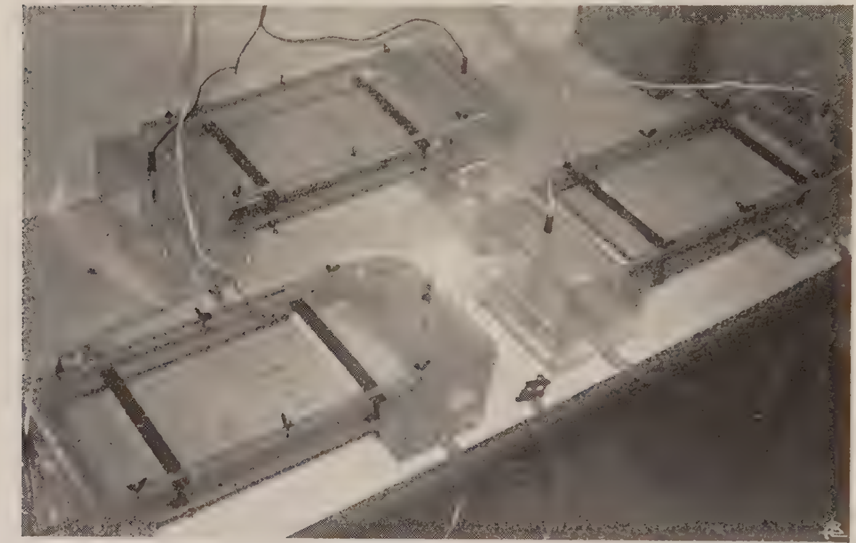


Fig. 4 — Apparatus for electrophoresis between glass plates

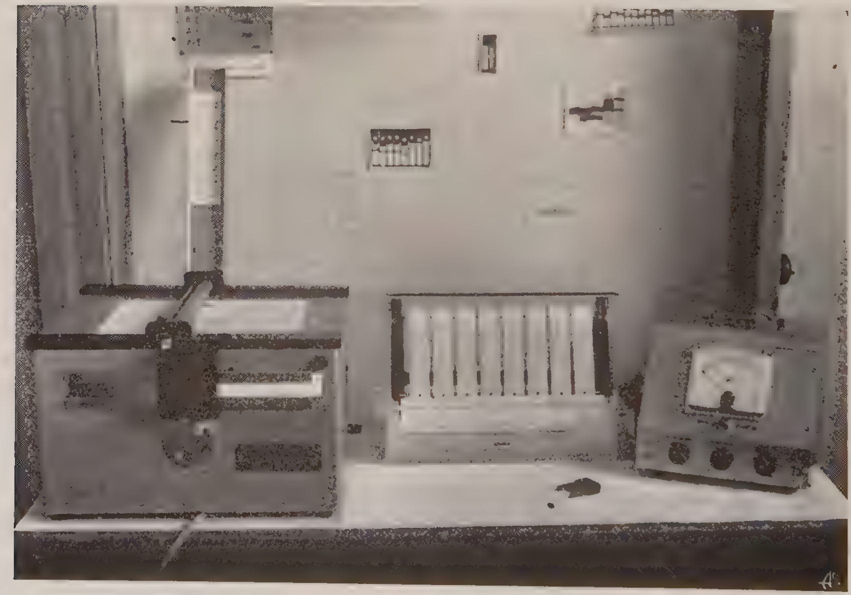
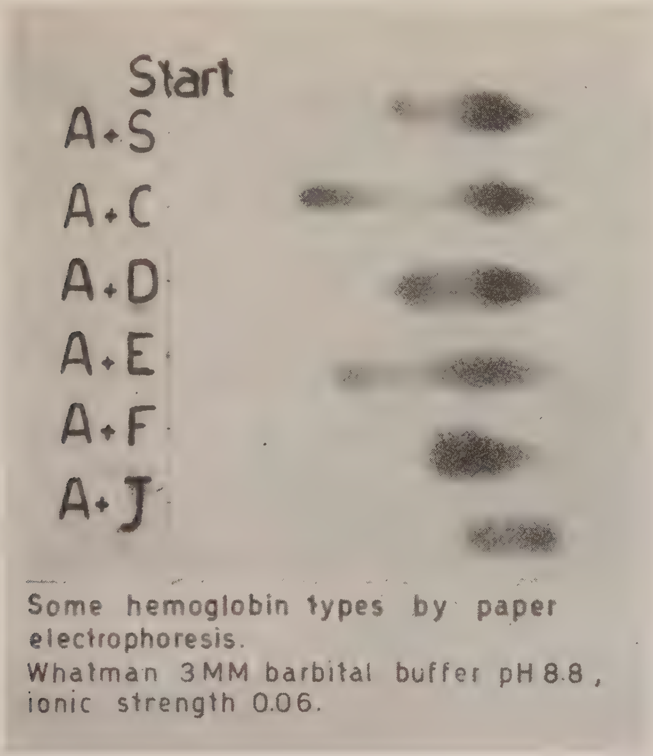


Fig. 5 — Apparatus for hanging strip electrophoresis with scanner

With these methods most of the now numerous abnormal haemoglobins have been discovered. The advantage of paper-electrophoresis is that the method is simple and that the number of samples that can be analyzed at the same time is considerable (Figs. 6 and 7).



Some hemoglobin types by paper electrophoresis.
Whatman 3MM barbital buffer pH 8.8, ionic strength 0.06.

Fig. 6

The limitations of the paper-electrophoretic techniques are that the results are only semi-quantitative and that small amounts of a certain fraction may easily escape notice. Moreover with these techniques it is only possible to get a very limited amount of a certain fraction in a pure state.

Fœtal haemoglobin has nearly the same mobility as normal adult haemoglobin; therefore these methods are not suited for the identification of fœtal haemoglobin.

Electrophoretical methods in other media, as starch, starch-gel and agar, have recently been worked out. They miss some of the limitations of paper-electrophoresis. The starch slab electrophoresis of Kunkel and Wallenius (8) has already found wide application. This method is especially suited to study the heterogenity of normal haemoglobin (A_1, A_2, A_3 fractions) (10, 11).

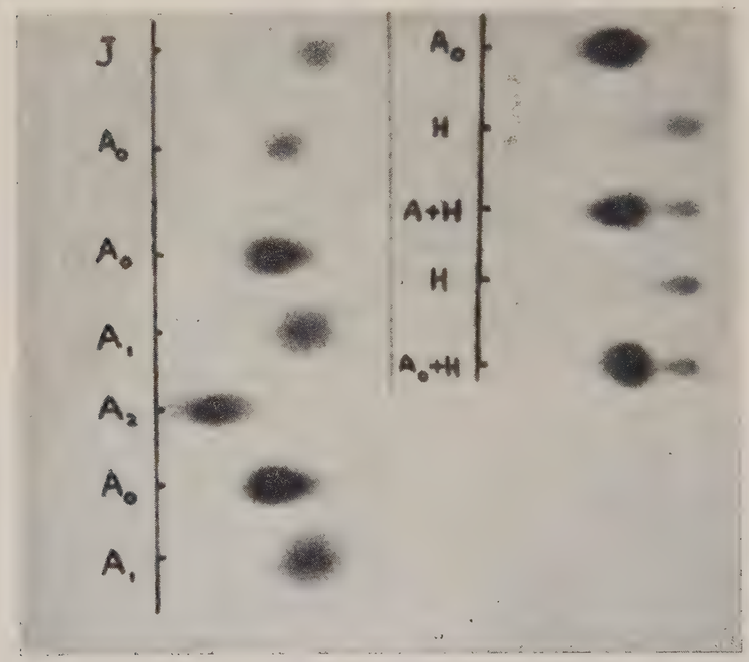


Fig. 7 — Paper electrophoresis of haemoglobin samples purified with carboxymethylcellulose chromatography

Starch electrophoresis, however, is more difficult to perform than paper electrophoresis and less suited for mass investigations (Fig. 8).

Agar electrophoresis has been described by Zuelzer and is applied for clinical purposes (12).

Starch-gel electrophoresis has been used for the separation of haemoglobin by Engle *et al.* (13).

Besides electrophoretical methods chromatographic techniques have recently been worked out. Good results have been obtained with column chromatography on Amberlite I.R.C.-50 and on carboxymethylcellulose. Amberlite I.R.C.-50 has been used first by Boardman and Partridge (14) and later by Prins and Huisman (15).

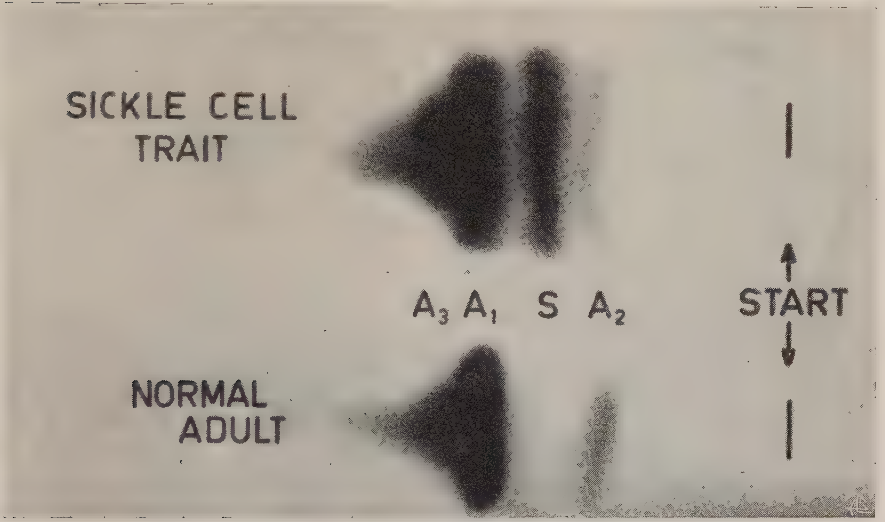


Fig. 8 — Starch slab electrophoresis

With the use of Amberlite I.R.C.-50 most abnormal haemoglobins and also fœtal haemoglobin can be separated from normal haemoglobin. Huisman and Prins (16) worked out a simple technique of Amberlite I.R.C.-50 chromatography in flat columns which is well suited for mass investigations (Figs. 9 and 10). For the latter purpose this method can be well used next to paper-electrophoresis (Fig. 11). Allen and Schroeder (17) recently described the separation of a minor component, possibly A_3, into 3 fractions and that of haemoglobin F into 2 components with a new form of chromatography on Amberlite I.R.C.-50.

Very promising results have been obtained with carboxymethylcellulose chromatography, first developed by Petersen and Sober (18).

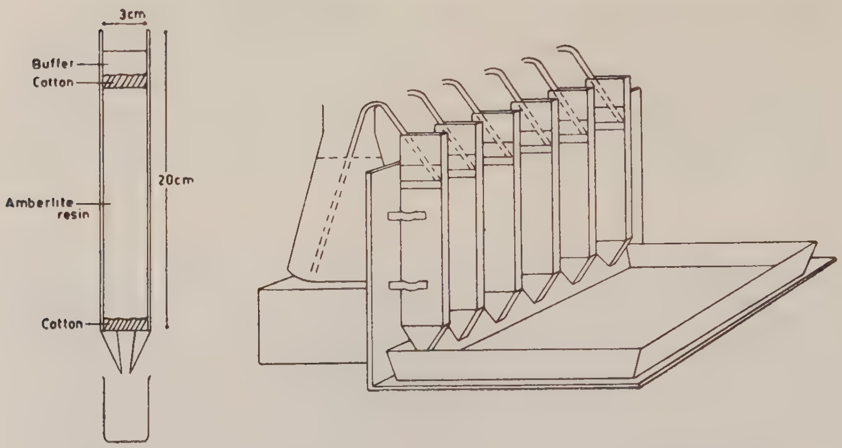
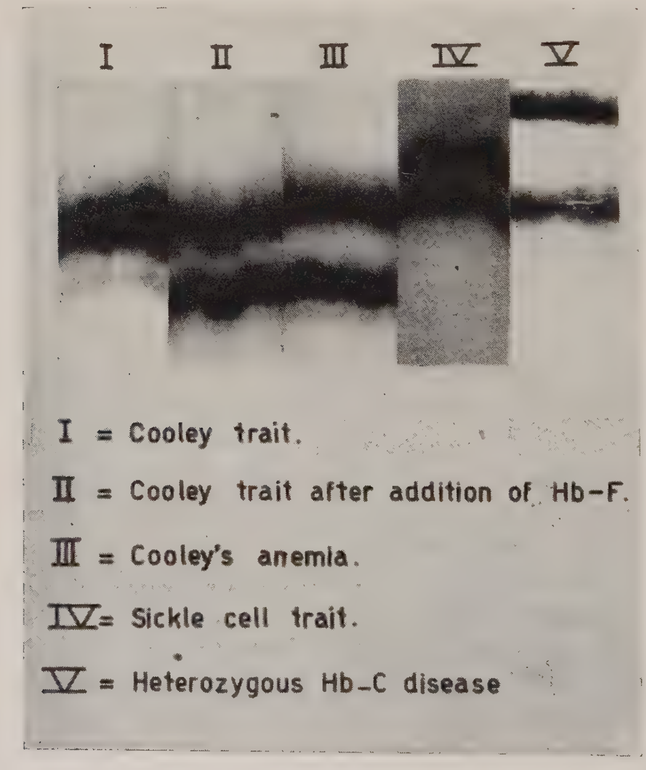

3cm
Buffer
Cotton
20cm
Amberlite resin
Cotton

Fig. 9 — Flat perplex cuvettes for Amberlite I.R.C.-50 chromatography



I = Cooley trait.

II = Cooley trait after addition of Hb-F.

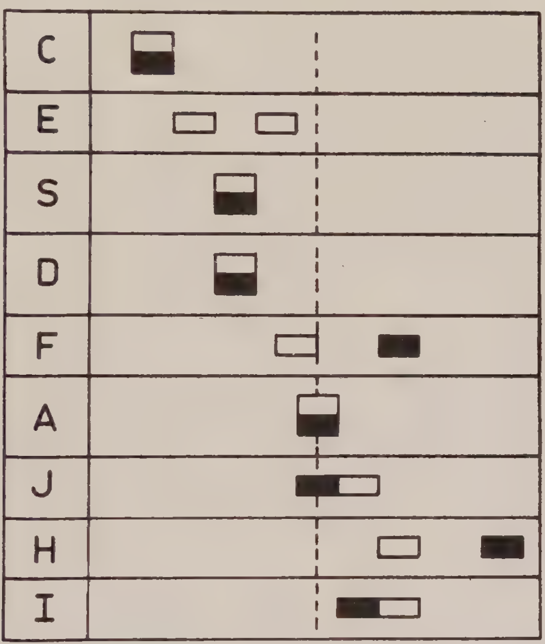
III = Cooley's anemia.

IV = Sickle cell trait.

V = Heterozygous Hb-C disease

Fig. 10 — Separation of different blood samples with Amberlite I.R.C.-50 chromatography

This method makes it possible to obtain the different fractions in a pure form in a somewhat greater quantity and is also well suited for quantitative work. Besides fœtal and abnormal haemoglobins the A_1, A_2 and A_3 fractions can be separated by this method (19). The method is, however, time consuming and therefore not so well suited for the normal routine laboratory (Figs. 12 and 13).



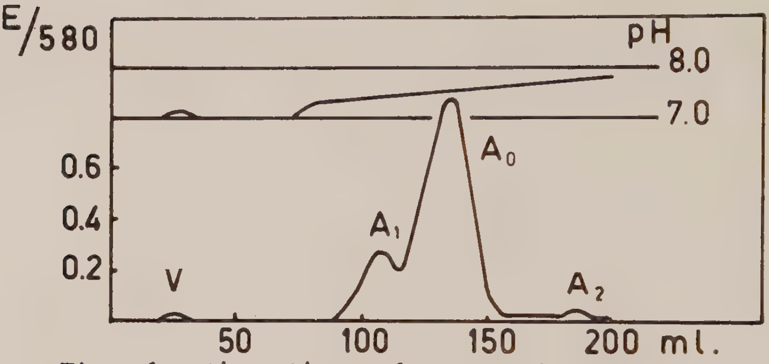
Comparison of the relative mobilities of different types of human hemoglobin in

paper electrophoresis ▭ and
chromatography ■

Fig. 11

There is a number of other possibilities to differentiate haemoglobins that should be kept in mind.

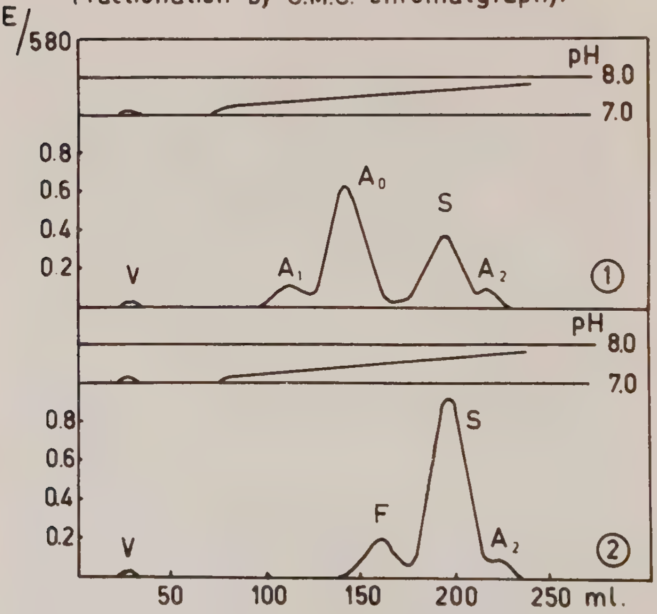
Fœtal and adult haemoglobin have a different solubility in phosphate solutions. The marked difference in solubility between



The fractionation of normal adult Hb by C.M.C. chromatography.

Fig. 12

Fractionation by C.M.C. chromatgraphy.



1. sickle-cell trait; 2. sickle-cell anemia.

Fig. 13

sickle-cell oxyhaemoglobin and sickle-cell haemoglobin is well known (Fig. 14). The difference in ultraviolet absorption spectrum between haemoglobin A and haemoglobin F may be used for the detection of the latter.

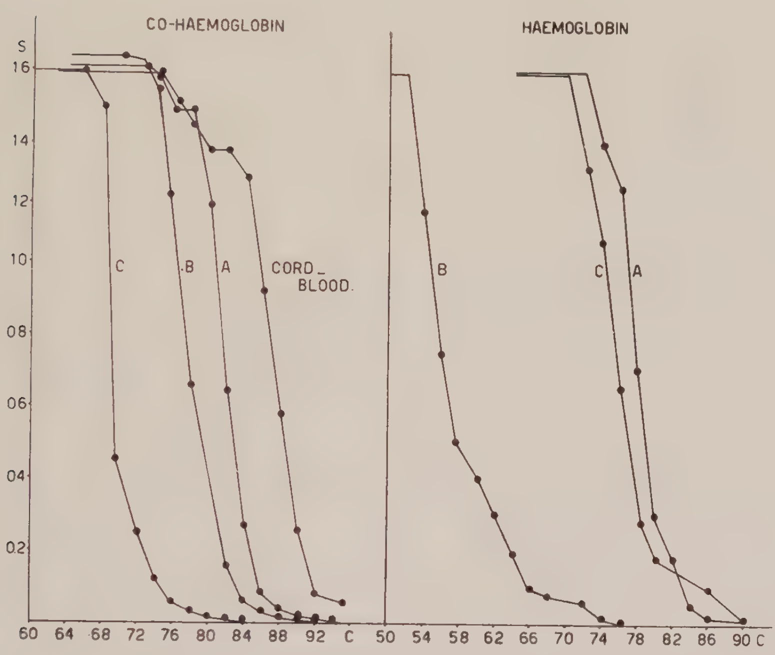


Fig. 14 — Solubility curves of different human haemoglobins. *Horizontal axis:* concentration of phospate-buffers solution in percent. *Vertical axis:* extinction of the filtrate at 540 mμ

Up till now no differences have been found in the oxygen dissociation curves of the various human haemoglobins. Contrary to what has been found earlier, even the dissociation curves of pure human adult and fœtal haemoglobin seem to be the same (20). In some mammals, however, differences in dissociation curves between the various haemoglobins (which have been found for instance in sheep) could be demonstrated (21) (Figs. 15 and 16). The possibility that in the human a special form of haemoglobin with a higher or lower affinity to oxygen exists, should not be ruled out.

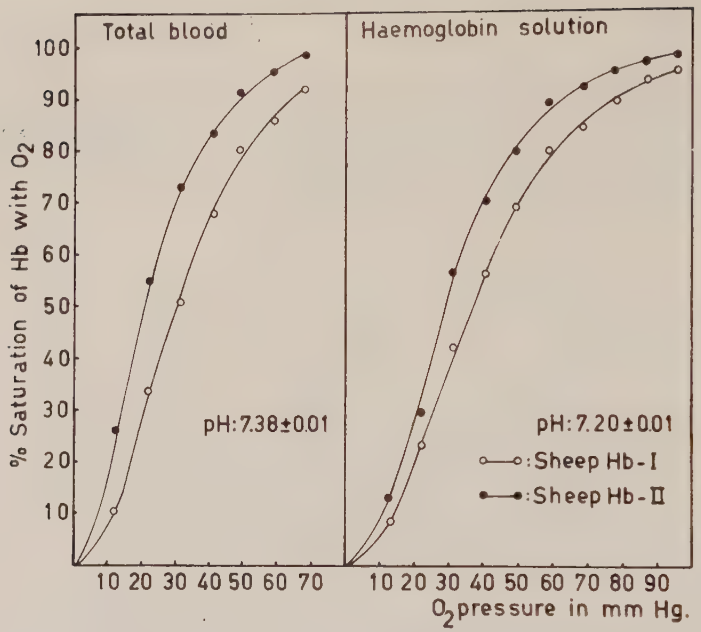


Fig. 15 — Oxygen dissociation curves of blood of sheep with Hb I or Hb II

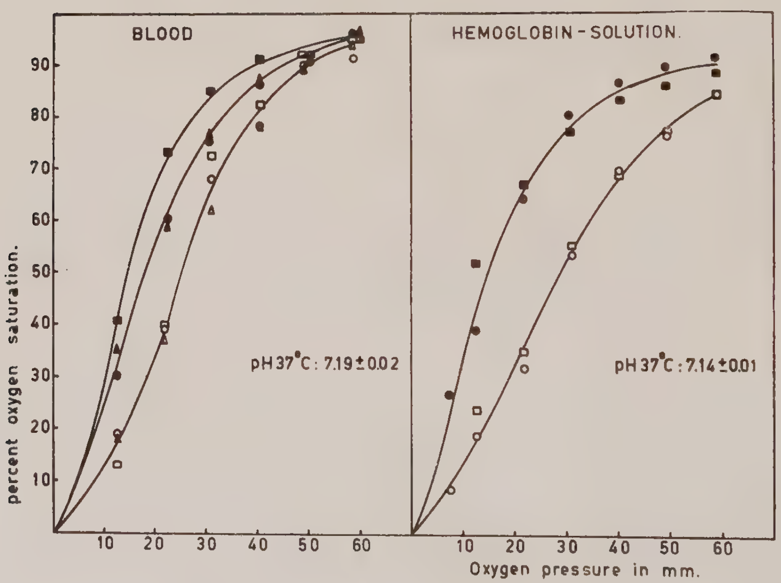


Fig. 16 — The oxygen dissociation curves of the blood of fetuses of the goat, of a newborn goat and of an adult goat

○—○ adul goat, in lamb for 91 days. Δ—Δ item for 115 days. □—□ item after delivery. ●—● fetus, 91 days after conception. ▲—▲ item after 115 days. ■—■ newborn goat

A laboratory manual with a survey of the most important methods has been published recently, as a result of the C.I.O.M.S. Symposium on abnormal haemoglobins, Istanbul 1957 (MacMillan, 1958).

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THE PATHOLOGY OF HEMOGLOBIN S

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PATHOLOGICAL PHYSIOLOGY

Sickle-cell or S hemoglobin is less soluble than is normal hemoglobin when in the reduced or deoxygenated state. Under conditions that favor hypoxia, the sickle-cell hemoglobin crystallizes. The elongated and pointed crystals distort the erythrocytes, producing sickled cells. The sickling of erythrocytes is associated with an increase in the viscosity of the blood and interference with its flow. Associated phenomena include hypoxic injury to the cells of the vessel walls, vasoconstriction followed by vasodilation, perivascular edema, hemoconcentration and the activation of thromboplastic substances with the production of intravascular clots. Occlusion of blood vessels due to combined factors leads to interference with the blood supply, tissue ischemia and necrosis (1, 2, 3, 4). Obstruction of blood flow in the cellular and fatty bone marrow may lead to necrosis and the mobilization of fragments of marrow and trabecular bone which escape into the afferent veins, producing fat and tissue emboli to the lungs and other viscera (5, 6, 7, 8).

The stasis of blood in capillaries and venous sinusoids over a period of years due to sickled erythrocytes, together with organization of the local areas of necrosis, leads to progressive replacement of the parenchyma by fibrous tissue.

Lesions due to vascular occlusion caused by agglomerations of sickled erythrocytes may occur in any organ of the body, but are most

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frequent in organs in which there is a sluggish flow of blood as in the spleen and in the bone marrow and in tissues in which there is a high oxygen uptake as in the gray matter of the brain and in the kidney. The degree and rapidity of sickling and the frequency and extent of obliterative lesions are proportional to the percentage of concentration of sickle-cell hemoglobin, being most marked in sickle-cell anemia, least marked in the sickle-cell trait and intermediate in hemoglobin S-C disease and in sickle-cell disease combined with thalassemia and other hereditary erythrocytic abnormalities.

In a given organ the lesion caused by occlusion of vessels is more likely to be in the portion which is the greatest distance from the arterial blood supply. Examples are ischemic necrosis of the head of the femur, hemorrhagic infarction of the cerebral cortex (Fig. 1), and papillary renal necrosis (Fig. 2). In the spleen there may be a layering of pathological lesions with the most marked fibrosis and iron deposition in the distal portion where the oxygen concentration is lowest (Fig. 3). In one of our patients, whose legs were amputated because of incurable leg ulcers, there was red marrow in the middle of the shaft of the tibia adjacent to the nutrient artery. The marrow was also relatively well preserved in the ends of the bone, but in the intermediate zone, between the sites of the more favored circulation, there was aseptic necrosis (Fig. 4). In the retina the vascular abnormalities are most frequently found in the periphery, rather than in the central portion (9, 10). In the intestinal tract the first lesions are ulcerations of the mucosa. Stasis of blood in the corpus cavernosum, with sustained and painful erection of the penis (priapism), is another example of lesions produced in any hypoxic area by the aggregation of sickled cells.

SICKLE-CELL TRAIT

In patients with hemoglobin A-S disease (sickle-cell trait) one finds at autopsy sickled erythrocytes in the capillaries of tissues fixed in formaldehyde. In the spleen there is pooling of the blood at the margins of the Malphigian bodies. The production of this lesion probably occurs at the time of or immediately preceding death, for there is no evidence of pigment deposition or siderofibrosis.

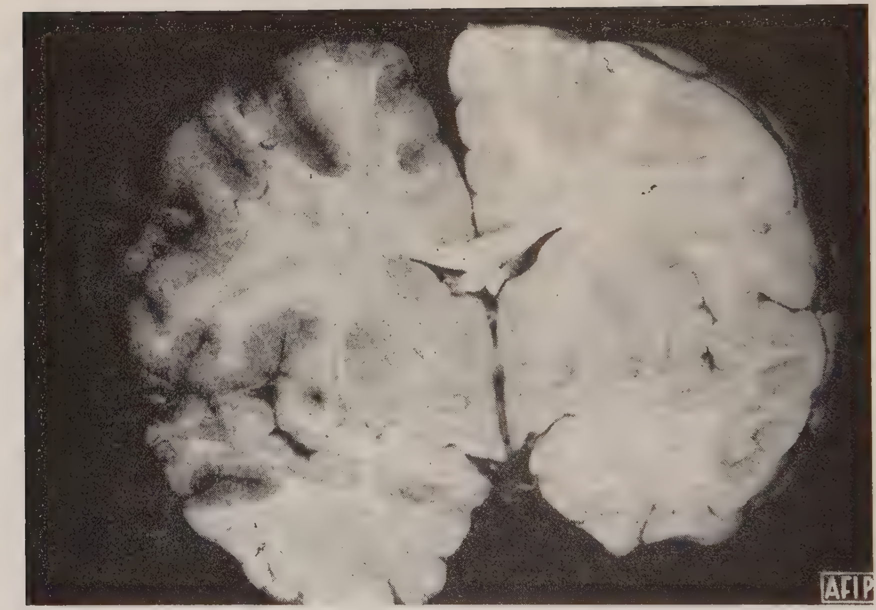

Fig. 1 — Sickle-cell anemia. Brain, unilateral hemorrhagic infarction of cerebral cortex. (A. F. I. P. Neg. No. 219650–200915)

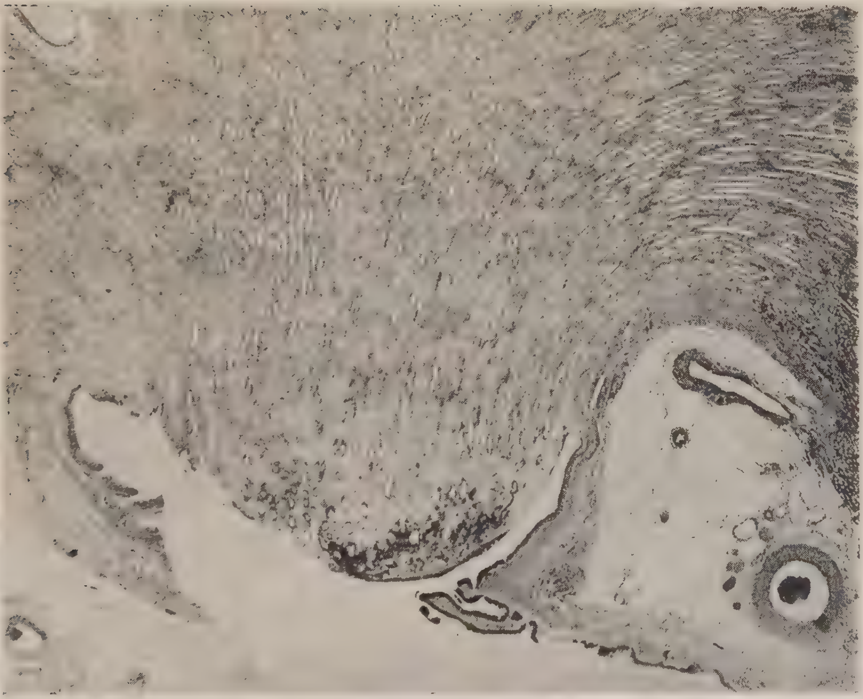

Fig. 2 — Sickle-cell trait. Hemorrhagic necrosis at tip of renal papilla. × 10. (A. F. I. P. Neg. No. 298389–03041)

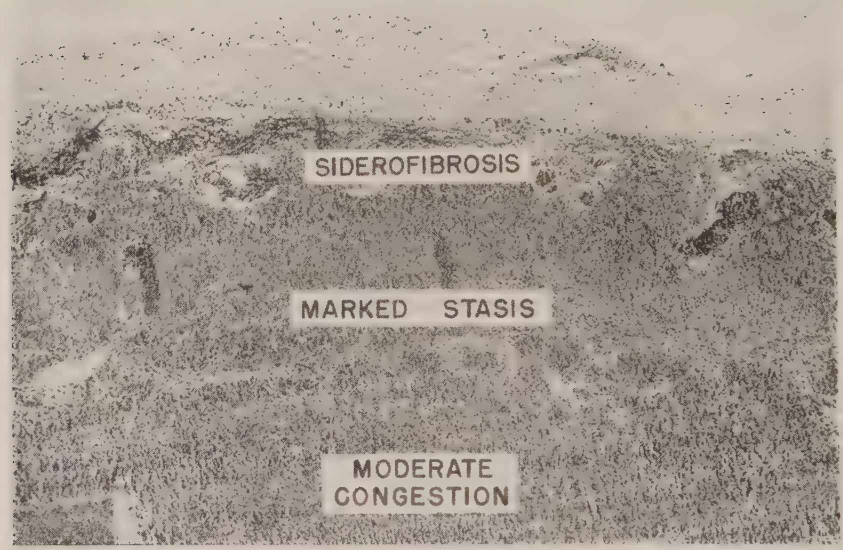
SIDEROFIBROSIS
MARKED STASIS
MODERATE CONGESTION

Fig. 3 — Spleen. Sickle-cell disease, probably hemoglobin S-C disease. Siderofibrosis in subcapsular area where hypoxia is most marked. In intermediate zone there is marked stasis with slight siderofibrosis. In the proximal zone in which there is better aeration of blood the congestion with sickled erythrocytes is moderate and there is minimal fibrosis

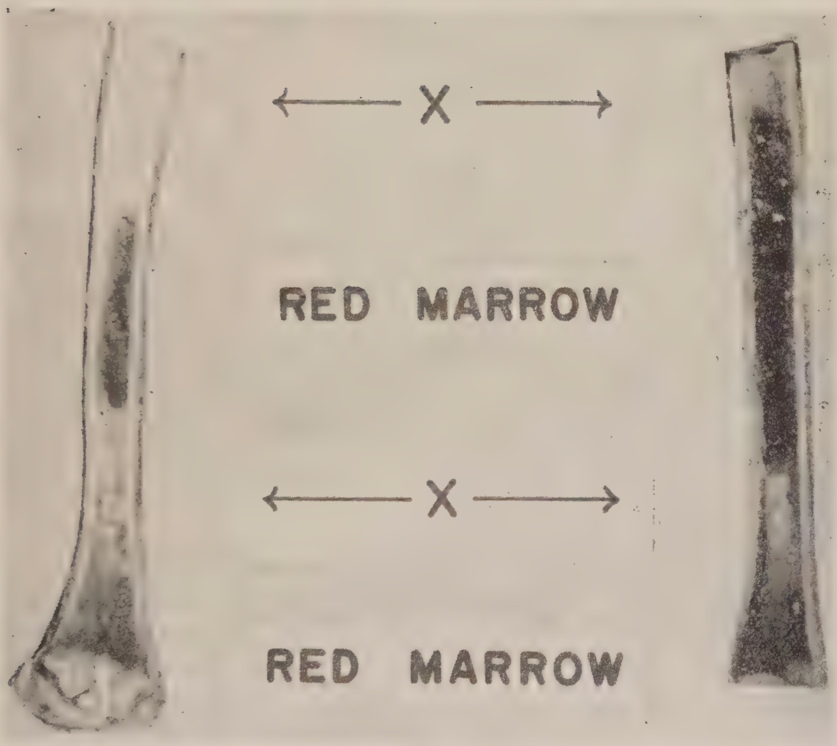

Fig. 4 — Sickle-cell anemia. Section of right and left tibiae. Necrosis of marrow at site of greatest hypoxia. Survival of red marrow at ends of bones and middle of shaft where the blood supply is more favorable

Patients with the sickle-cell trait are more prone to develop complications and to succumb to various diseases associated with circulatory stasis than are normal individuals. Often at autopsy there are focal areas of infarction and necrosis associated with and probably caused by the presence of sickled cells superimposed on a primary factor such as heart failure, vasospasm, infectious agent or tumor.

In rare instances, patients with sickle-cell disease of the A-S type may have a fatal illness characterized by an acute onset with severe headache, muscle pains, petechiae and symptoms and signs referable to the central nervous system. At autopsy these patients have evidence of arteriolar occlusive vascular disease and multiple hemorrhagic infarcts in the white matter of the brain (7, 11).

Massive congestion of the spleen with varying degrees of splenic infarction may occur in patients with the sickle-cell trait who are exposed to the low oxygen concentrations during airplane flights (12, 13, 14).

Unilateral hematuria, which occurs in a small percentage of patients with hemoglobin A-S disease, is due to the stasis caused by sickled red cells in the distal tubular area producing papillary renal necrosis (15) (Fig. 2). The bleeding is usually from the left kidney which is explained by the fact that the left renal vessels are longer and more tortuous than those on the right.

Frequent instances of sudden and unexpected death have been observed in patients with the sickle-cell trait (16). Alcoholic inebriation, traumatic shock, sedatives and anesthetics or other conditions favoring hypoxia are apparent trigger mechanisms. At autopsy these patients have a large spleen which resembles a «bag of blood». There is marked engorgement of the sinusoids of the liver and other viscera, dilatation of the right heart, sickle-cell aggregates in the pulmonary arterioles and in some cases small areas of focal necrosis in the liver, brain and other organs.

SICKLE-CELL ANEMIA

In patients with sickle-cell anemia (Hgb S-S) there are few anatomical lesions other than sickled erythrocytes during the first months of life. As the fetal hemoglobin, which is relatively soluble in the deoxygenated state, is replaced by sickle-cell hemoglobin which readily crystallizes, the disease becomes manifest. Among the first abnormalities to appear are congestive enlargement of the liver and spleen, dactylitis (Fig. 5) and anemia of the hemolytic type associated with acholuric jaundice.

The spleen in sickle-cell anemia undergoes a series of progressive changes from congestive enlargement to fibrotic atrophy (17). Autopsies of infants and young children with sickle-cell anemia reveal marked congestion of the spleen which is maximal within and adjacent to the Malpighian bodies where the blood, under arterial pressure, meets with the obstruction caused by the sickled cells entangled in the splenic pulp. Organization of extravasated blood in areas of chronic stasis leads to the formation of siderofibrotic nodules (Gamna-Gandy bodies) which are characterized by pigment granules and crystals, pigment-laden macrophages, foreign body giant cells, fibrosis and calcium and iron depositions (Fig. 6). In older patients there is gross distortion of anatomical pattern. Infarcts in various stages of healing may be demonstrable. The capsule is thickened and wrinkled. In adults the spleen is usually a shrunken remnant consisting of a few thickened blood vessels terminating in scar tissue.

In younger patients with sickle-cell anemia the marrow is hyperplastic and there is a widening of the marrow cavity with thinning of the cortex. There may be osteoporosis of the skull and in occasional patients, hair-on-end trabeculations. Infarctions of the red marrow of the fingers and toes are associated with pain and swelling (Fig. 5) and striking alterations in bony architecture. In older children the distal bones of the extremities are replaced by fibrous tissue and dactylitis is seldom observed. The red marrow of the long bones is congested and there is patchy necrosis, infarction and fibrosis, often with irregular thickening of the cortical bone and new bone formation within the marrow cavity. The trabecular pattern is distorted and there may be periosteal elevation. The bodies of the vertebrae tend to be flattened and compressed with cupping due to the pressure of the intervertebral discs. Aseptic necrosis of the head of the femur and of the humerus is common.

In localized areas the islands of hyperplastic marrow appear radiolucent in roentgenograms and simulate the «punched out» lesions which are produced by plasma cell myeloma and metastatic tumors.

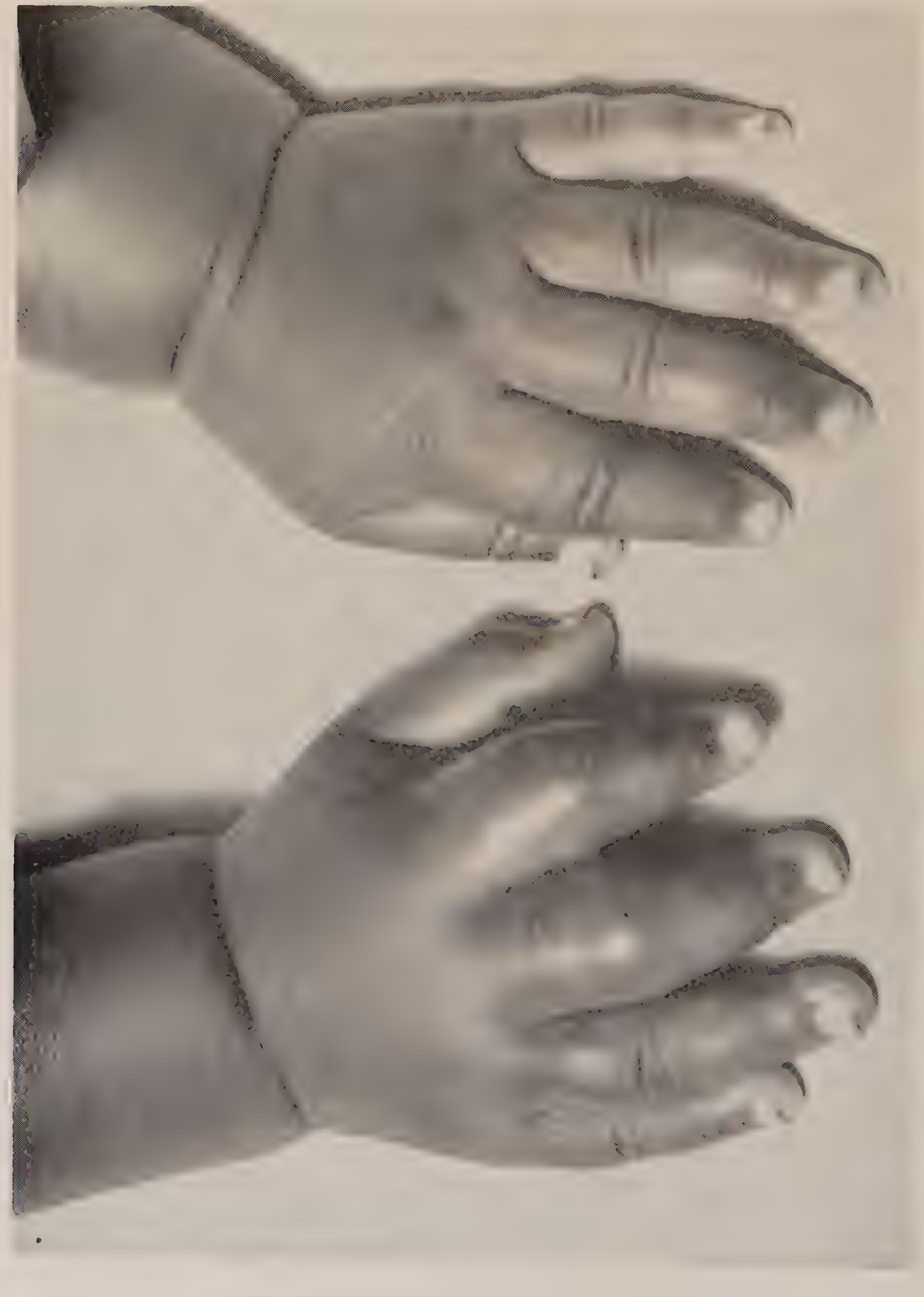

Fig. 5 — Sickle-cell anemia. Dactylitis due to infarction of bones of fingers

Smith and Conley (18) and Prowler and Smith (19) have described marked translucency, localized areas of opacity and abnormal trabecular patterns in dental films. Osteomyelitis of the bones is relatively common. The bones are particularly susceptible to infections whith *salmonella* organisms, which may produce extensive destruction of bones in multiple sites.

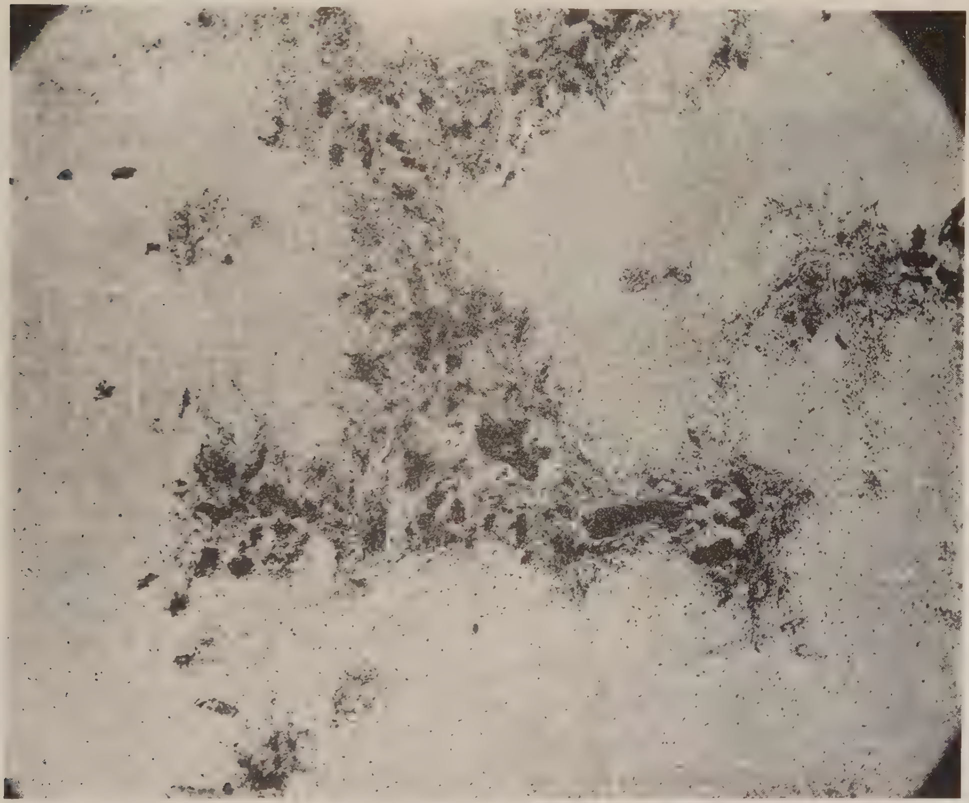

Fig. 6 — Spleen, sickle-cell anemia. Congestion of pulp with sickled erythrocytes. Siderofibrotic nodules. (A. F. I. P. Neg. No. 5613014-712840)

In the brain, the most common lesion is a hemorrhagic infarction that is produced by sickling of red cells in the capillaries of the gray matter and meninges. Thrombosis of the meningeal and dural veins may be demonstrable. The lesions are often unilateral, producing hemiplegias in young individuals. Because of the uneven distribution of the vascular lesions in the central nervous system due to sickle cells, there may be bizarre neurological and mental signs and symptoms.

The liver in sickle-cell anemia is characteristically enlarged. There is marked congestion of the sinusoids with sickle cells. Fibrin networks and hyaline thrombi are demonstrable. The Kupfer cells may contain many phagocytized red blood cells and blood pigment. Hemosiderin is present in the liver cord cells. In patients dying during crises, there may be focal necrosis of the liver and sometimes gross infarction. There may be intense jaundice due to circulatory stasis and injury to the liver cells (20, 21). This jaundice is of a regurgitant (obstructive) type. In older patients there may be fibrotic, nodular cirrhotic and atrophic changes in the liver. Gall stones are common.

The lung is the organ of the body which is best aerated, which is in constant movement and which has a rich blood supply. One therefore would not expect this organ to be involved in obliterative vascular disease. In practice one frequently sees patients with pulmonary symptoms and signs, including chest pain, cough, hemoptysis and roentgenological evidence of atelectasis and infarction. At autopsy, emboli in various stages of organization are demonstrable. In the lung the emboli are in the pulmonary arterioles whereas in all other organs of the body the luminal occlusions due to aggregated sickled cells and thrombi are usually in capillaries and veins. It is apparent that masses of agglomerated sickled cells and aggregates of sickled cells mixed with fibrin and with gels of hemoglobin crystals form in venous channels anywhere in the body. These dislodge, go to the lung, and there occlude the arteries (Fig. 7, A, B). They may cause wedge shaped infarcts involving major portions of the lung, but as a rule the collateral circulation is adequate to prevent major parenchymal injury. The cell aggregates and intravascular clots which primarily form in the lumens of the blood vessels become attached to the endothelium as plaques which later become organized and recanalized (Fig. 7, C, D). If the lesions are seen after organization has been completed, one is likely to interpret the finding as due to primary vascular proliferative disease or pulmonary arteriolar sclerosis (22). The true sequence of events is embolus, intimal injury and thrombus formation followed by organization and recanalization of the clot. Obliterative arteriolar lesions may be extensive and cause pulmonary hypertension and cor pulmonale.

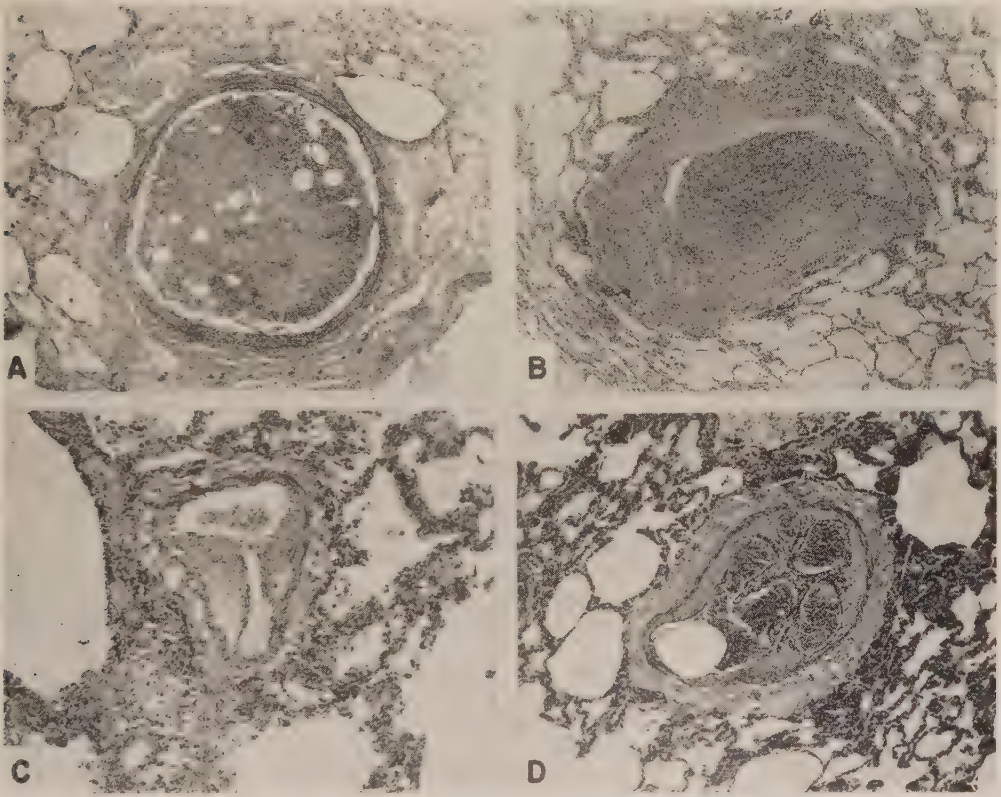

Fig. 7 — Sickle-cell anemia, lung: A, occlusion of arteriole by aggregate composed of sickled erythrocytes, leukocytes, fat globules and fibrin; B, arteriolar plaque, secondary to embolus; C, arteriolar plaque probably due to organization of embolus; D, recanalization of arteriolar embolus and thrombus

Other pathological lesions which have been observed include hemorrhagic infarction of the intestinal mucosa, pancreas and placenta. Chronic ulcers of the lower legs and ankle region are common in older patients.

Throughout the life of the patient with sickle-cell anemia there is decreased survival of erythrocytes. There is chronic unremitting anemia and signs of increased blood destruction including jaundice of the hemolytic type and iron-containing pigment in the phagocytes and in the tubular cells of the kidneys. There is compensatory hyperplasia of the bone marrow, with red bone marrow in the long bones of the body as well as in the flat and warm bones.

The usual crisis in sickle-cell anemia which is characterized by pain and fever is a vascular obliterative crisis and is not associated with a marked drop in the red cell values or necessarily with an increase in the degree of jaundice or in the pigment output in the feces and urine. There may be episodes of aplastic crisis.

COMBINATION DISEASES

In sickle-cell hemoglobin C disease the lesions are similar to those found in hemoglobin S-S disease but tend to be less advanced. The spleen of older patients with hemoglobin S-C disease may be small and atrophic as in sickle-cell anemia, but usually the spleen in S-C disease is enlarged and shows intermediate degrees of siderofibrosis. Patients with hemoglobin S-C disease often develop hematuria and aseptic necrosis of the head of the femur. Compression fractures of the vertebrae, chronic leg ulcers, retinal lesions and priapism have been described in hemoglobin S-C disease.

In sickle-cell-thalassemia disease the anatomical lesions are quite variable, but resemble, in general, those found in sickle-cell anemia.

SUMMARY

The tissue changes observed in patients with abnormal hemoglobin of the sickle-cell type are explainable on the basis of vascular occlusion caused by the formation of long, rigid and pointed crystals of hemoglobin. In patients with the sickle-cell trait without anemia (Hgb A-S) the vascular occlusive phenomena are minimal as a rule, but there may be unilateral hematuria, massive congestion and infarction of the spleen in association with high-altitude flying and sudden and unexpected death in association with inebriation and other conditions characterized by hypoxia. In sickle-cell anemia (Hgb S-S), there are few lesions during the first few months of life, but in later years there is unremitting impairment of blood

flow with gradual replacement of parenchyma by fibrous tissue. The spleen is most consistently and characteristically affected, the changes being those of congestion of splenic pulp, follicular hemorrhages, siderofibrosis and atrophy. Infarcts and local areas of tissue necrosis or scarring may occur in any organ, including the bone marrow, brain and cord, kidney, liver, lymph nodes, endocrine organs and gastrointestinal tract. Emboli may be demonstrable in the lung and occasionally may be extensive enough to cause pulmonary hypertension and cor pulmonale. Other characteristic lesions include aseptic necrosis of head of femur and humerus, occlusive vascular lesions in periphery of eyegrounds, priapism, papillary renal necrosis, leg ulcers, gall stones and placental infarcts. Pathological lesions in hemoglobin S-C disease and in thalassemia-sickle-cell disease are similar to those in homozygous sickle-cell anemia.

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HAEMOGLOBIN E

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Haemoglobin E was discovered in 1954 simultaneously by Itano *et al.* in the U.S.A. in a family of part Indian origin and by Chernoff *et al.* in Thailand in the Siamese.

PHYSICO-CHEMICAL PROPERTIES

Hb E is determined by its electrophoretic and chromatographic properties. On electrophoresis at pH 8.6 it has a mobility lower than Hb S and O, but is slightly faster than Hb C.

It has the same mobility as Hb A2. At pH 6.5 it is faster than Hb A, but is slightly slower than Hb S. On ion exchange chromatography using Amberlite IRC 50, it moves between Hb A and S.

GENETIC BASIS

The production of Hb E is under genetic control.

The gene for Hb E is inherited as a Mendelian dominant.

It is probably an allele of Hb A and probably not an allele of the gene for thalassaemia. Four hereditary conditions associated with Hb E have been recognised.

1. Hb E trait is the heterozygous condition.
2. Hb E-thalassaemia disease results from the combined heterozygous states for Hb E gene and the thalassaemia gene.

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 457-473, 1959

3. Hb E-disease is the homozygous condition.
4. Hb S-Hb E disease is the double heterozygous state for the gene for Hb S and for Hb E.

It has been shown that Hb E, although inherited, is formed at some time after birth (Lie-Injo Luan Eng, 1956*b*).

CLINICAL AND HAEMATOLOGICAL DATA

HAEMOGLOBIN E TRAIT.

Haemoglobin analysis in this condition shows the pattern Hb A + E, the pathologic Hb E being the minor component. The trait form is asymptomatic, but microcytosis of the erythrocytes and an increased resistance of the erythrocytes to hypotonic saline solutions may be present in some individuals (Chernoff *et al.*, 1956; Lie-Injo Luan Eng, 1956*a*). That these changes were not due to diseases prevalent in the Orient or to iron deficiency was shown in our unpublished study of healthy Hb E trait carriers. Intensive iron therapy in three of these carriers whose erythrocytes showed microcytosis and an increased resistance to hypotonic saline solutions did not alter these blood abnormalities.

As Hb E has the same mobility as Hb A2 and as the Hb A2 may be increased in thalassaemics, a thalassaemic may be mistaken electrophoretically for a Hb E trait carrier. However the amount of Hb A2 in thalassaemics is usually less than 15 %, while the Hb E component in Hb E trait carriers is in general higher than 20 % (Aksoy *et al.*, 1957).

HB E-THALASSAEMIA DISEASE.

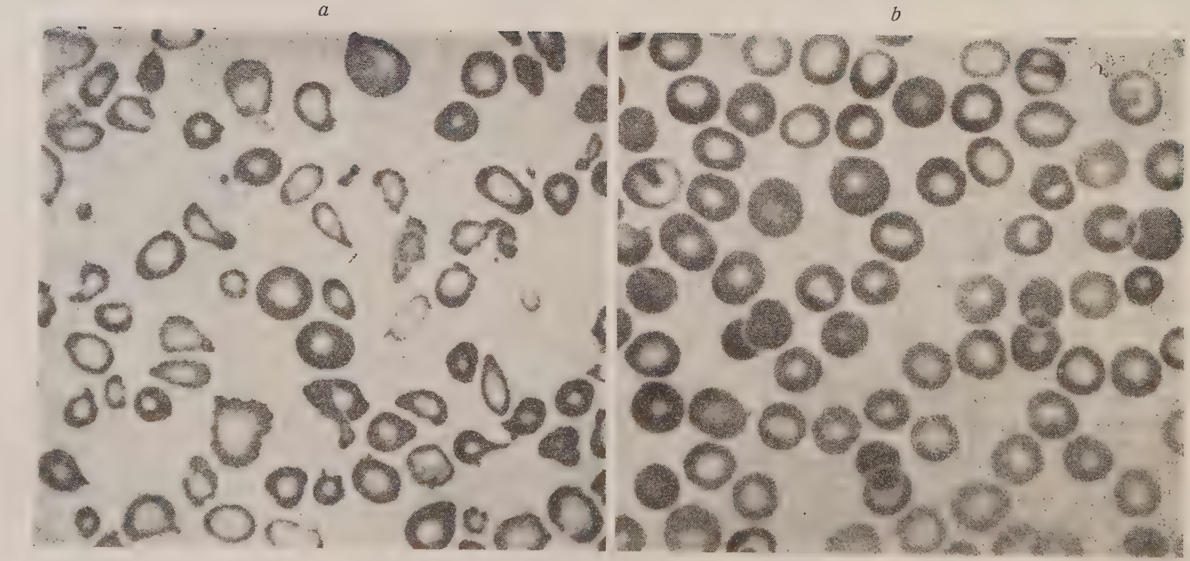
Hb E-thalassaemia disease is found more often in children. In three series where a significant number of cases were studied (32 cases described from Thailand by Chernoff *et al.*, 1956, 20 cases from India by Chatterjea *et al.*, 1957, and 30 cases from Indonesia, of which 20 cases published in 1957 by Lie-Injo), 62 were children, while 20 were over 12 years of age. From the description of Hb

E-thalassaemia disease in various countries (Thailand, Indonesia, Cambodia, Malaya, India and Ceylon) it can be concluded that the clinical manifestations of this disease are most variable. One may classify these cases into severe, moderately severe and mild. This classification, however, is entirely artificial as the groups merge.

a) In the severe Hb E-thalassaemia disease group, all patients suffered from severe haemolytic anaemia and symptoms due to the anaemic condition with impaired health and poor development. The symptoms develop early in infancy. The spleen and liver are nearly always enlarged, the heart is often enlarged with systolic murmurs. The haematologic findings are indistinguishable from those found in severe cases of thalassaemia major. The anaemia is usually microcytic hypochromic with marked aniso-poikilocytosis and polychromasia of the peripheral blood (Fig. 1, *a*). Target cells, basophilic stippeling and normoblasts are seen. Howell Jolly bodies and Cabots rings may be found. Reticulocyte count is elevated; the number of thrombocytes is usually normal, but may sometimes be slightly reduced. The leucocyte count is often elevated frequently with a moderate shift to the left on differential count. Osmotic fragility of the erythrocytes to hypotonic saline solutions was decreased in all of the cases examined. The indirect reacting serum bilirubin is increased. The bone marrow is usually hyperactive normoblastic. In several cases Chatterjea (1957) found megaloblasts in the bone marrow, probably caused by associated nutritional deficiency. Red cell survival studies revealed significantly decreased life spans of the erythrocytes (Chernoff *et al.*, 1956). Haemoglobin analysis in severe cases showed the pattern E + F or E + F + A, Hb E being the major component. The amount of Hb F in the blood varies considerably.

Röntgenologic changes of the bones are regularly seen, resembling those found in thalassaemia major (Fig. 2).

b) The moderately severe cases enjoy moderately good health and are more active. The anaemia is moderately severe and the symptoms are milder. However, it should be stressed that the severity of a case may vary at times, and that the severity of cases in the same family may differ considerably. Pregnancy can greatly influence the anaemic condition. In one of our moderately severe cases the anaemic



(880 ×) (930 ×)

Fig. 1 — Peripheral blood smear in Hb E-thalassaemia disease; *a*, extreme derangement of the red cell morphology in severe Hb E-thalassaemia disease in a child; *b*, only slight changes in red cell morphology in an adult case without anaemia but with 45 % foetal haemoglobin in his red cells

condition was aggravated by each pregnancy, leading to repeated premature birth (Lie-Injo Luan Eng *et al.*).

c) In the milder form the patients do not complain of illness, lead an active live and may not be anaemic.

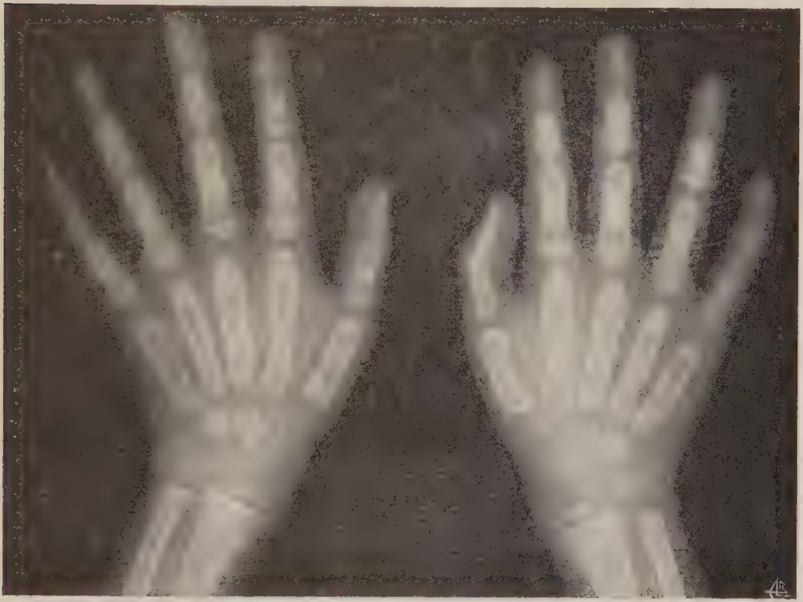


Fig. 2 — Bone changes in the hands of a child 5 years of age with Hb E-thalassaemia disease. Note cortical thinning, osteoporosis with coarse trabeculation and rectangular contour of the metacarpals. The changes are indistinguishable from those found in thalassaemia major

One of our cases in the mild group had a haemoglobin level between 13.3 g to 14 g %. He had, however, about 45 % fœtal haemoglobin in his red blood cells and a slightly enlarged spleen. The red cell morphology of his peripheral blood was only slightly deranged (Fig. 1, *b*). Another case, a woman with a haemoglobin 12 g % was healthy. She had a haemoglobin pattern of E + A with Hb E being the major component, while the amount of fœtal haemoglobin was not increased. One of her children suffered from severe thalassaemia major (Lie-Injo Luan Eng, 1956*a*, 1957). Such extremely mild cases of Hb E-thalassaemia disease without anaemia have not been described by others as far as we know.

HB E-DISEASE (homozygous condition).

All the cases of Hb E-disease, 6 described by Chernoff *et al.* (1956), 2 by Lehmann *et al.* (1956), 3 by ourselves (Lie-Injo Luan Eng & Oey Hoey Giok, 1957) and later 5 additional cases found by us, one by Brumpt *et al.* (1956), and 5 by Na-Nakorn and Minnich (1957) were mild. The 22 cases mentioned above were diagnosed by finding on electrophoresis one spot consisting of Hb E, while the amount of Hb F present was not or was only slightly elevated (up to 8 %). In only 8 cases (2 described by us, one by Brumpt *et al.* and 5 by Na-Nakorn and Minnich) were both parents examined, and were both found to have Hb E in the blood. Physical symptoms in this disease are unremarkable, except that the spleen or liver or both may be slightly enlarged. In 5 cases (the 2 of Lehmann and 3 of ours) there was no anaemia, and these people were healthy and led an active life. The others had a mild anaemia. In several of our slightly anaemic cases it was noticed that accompanying diseases may cause the haemoglobin level to drop temporarily. Microcytosis of the red blood cells is a constant finding with a normal M.C.H.C. In some patients an unusually high erythrocyte count was found. The red cell morphology has been described as being characterized by a striking number of target cells, otherwise the red cell morphology is only slightly deranged (Fig. 3, *a*). In the Indonesian series target cells were not always abundant (Lie-Injo Luan Eng & Oey Hoey Giok, 1957) and in two of the cases very few target cells were seen (Fig. 3, *b*). The resistance of the erythrocytes to hypotonic saline solutions was always increased. Signs of increased haemolysis, such as raised serum bilirubin, an increased urinary urobilinogen, slightly elevated reticulocyte count or an increase of the erythrocytic elements in the bone marrow, may occasionally be found. In one of the patients of Na-Nakorn and Minnich a slightly shortened red cell survival time was found. No bone changes have been detected in 13 cases examined radiologically. As mild cases of Hb E-thalassaemia disease may resemble cases of homozygous haemoglobin E disease, and laboratory data for the two conditions are overlapping, the only way

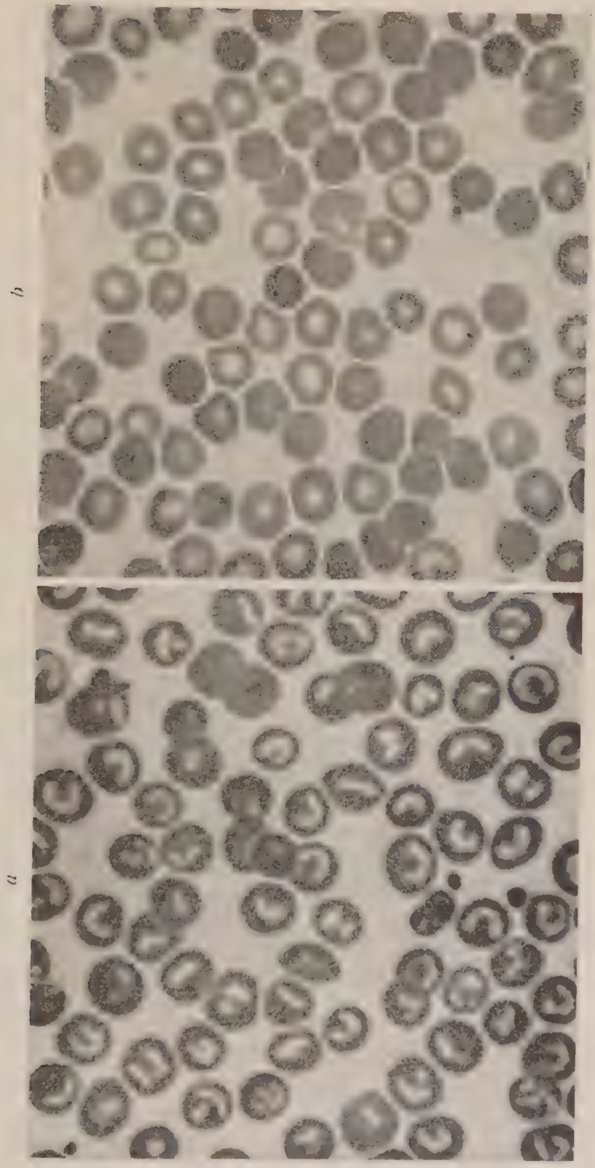


Fig. 3 — Peripheral blood smear in homozygous Hb E disease: *a*, a case with numerous target cells; *b*, a case with very few target cells. Both cases had no anaemia. (930×).

to differentiate those two conditions with certainty is an adequate family study in addition to the laboratory examinations.

In addition to the cases mentioned above, several other cases of homozygous Hb E disease have been encountered electrophoretically during surveys (Brumpt *et al.*, 1957, Lehmann and Singh, 1956, Chatterjea, 1957, Vella, 1958), but no clinical and haematological data are available.

HB S-HB E DISEASE.

Aksoy and Lehmann (1957) described in an Eti-Turk family the only 2 cases of Hb S-Hb E disease known. These two cases, a mother of 70 years old and her son, had a mild hypochromic anaemia. In the mother the haemoglobin consisted of 60 % Hb S and 40 % Hb E. She had occasional joint and bone pains and suffered from a myocardial infarct. Mild radiologic changes were found in the bones. Her son had a slightly enlarged spleen.

Therapy.

There is no causal therapy for anaemia in Hb E-thalassaemia and Hb E disease. The only effective therapy is blood transfusion when anaemia is severe. As regards splenectomy, in cases of Hb E-thalassaemia disease, it is too early to evaluate the value of this procedure, but it seems that it has been of benefit in certain cases.

DISTRIBUTION

Hb E is found mainly in South East Asian countries. In Thailand the incidence ranges from 8 to 32.7 % (Na-Nakorn *et al.*, 1956). In Cambodia an incidence of 37 % among patients and 28.8 % among normal persons was reported (Brumpt *et al.*, 1957). In Burma the incidence is 15.3 % (Lehmann *et al.*, 1956). In Malaya it is 7,3 % (Lehmann and Singh, 1956), while in Singapore 5.19 % of the Malay population carries Hb E (Vella, 1958). In Indonesia the incidence of Hb E varies considerably in various ethnic groups, from none to 20 %, the highest incidence being in people from the lesser

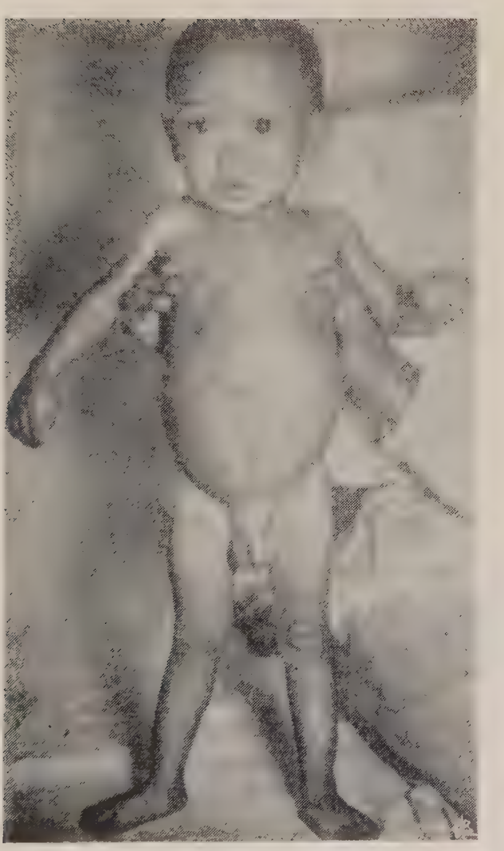


Fig. 4 — Severe Hb E-thalassaemia disease in a boy 1 1/2 year old showing dystrophia, hepato-splenomegaly

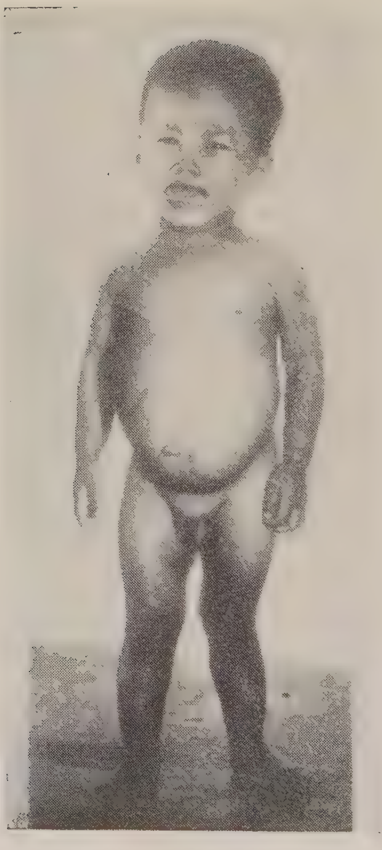


Fig. 5 – A case of moderately severe Hb E-thalassaemia disease. The boy is ± 2 years old and is in a rather good nutritional state

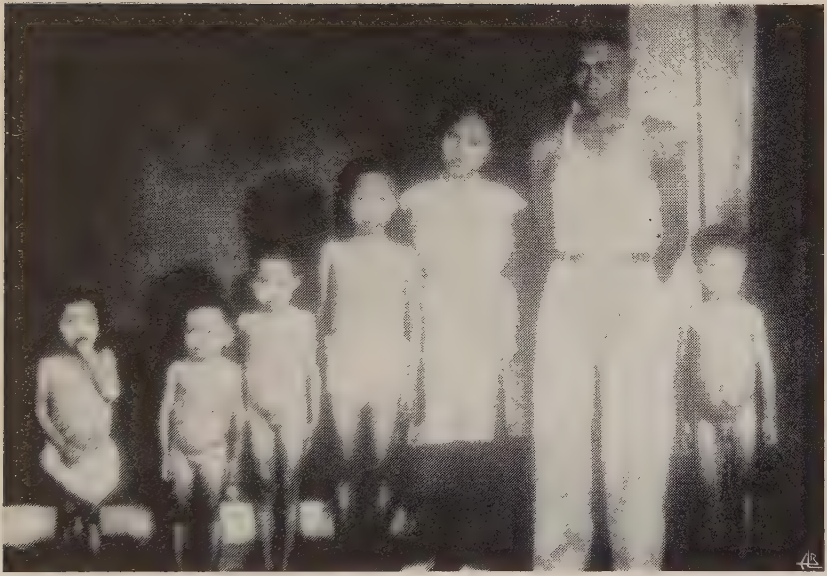


Fig. 6 — Several cases of Hb E-thalassaemia disease who came for a check up. See difference in age. The girl is 13 years old and the man is 35 years old

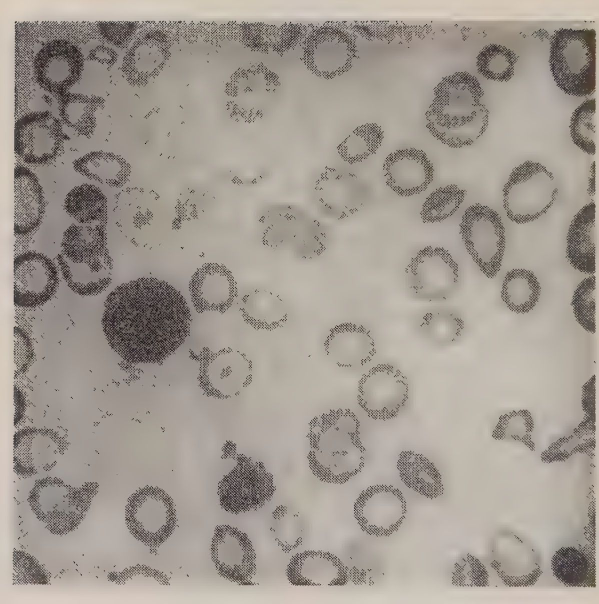


Fig. 7 — Peripheral blood in a case of moderately severe Hb E-thalassaemia disease

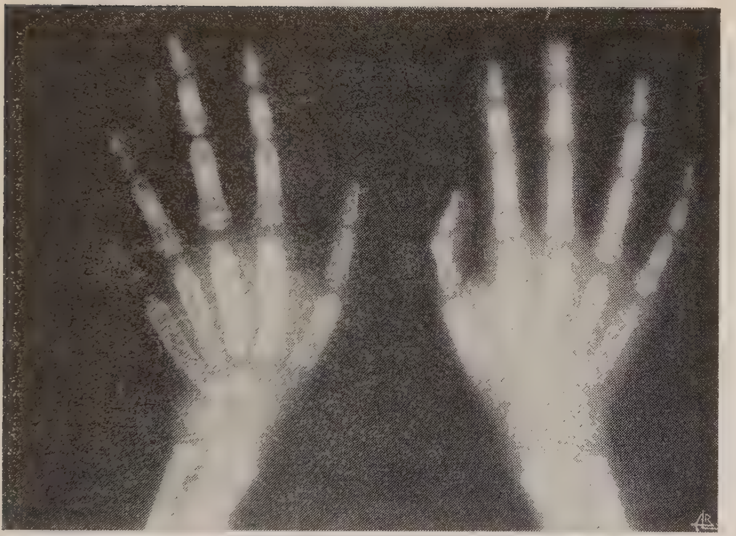


Fig. 8 — Bone changes in the hands of a child with Hb E-thalassaemia disease resembling those found in thalassaemia major

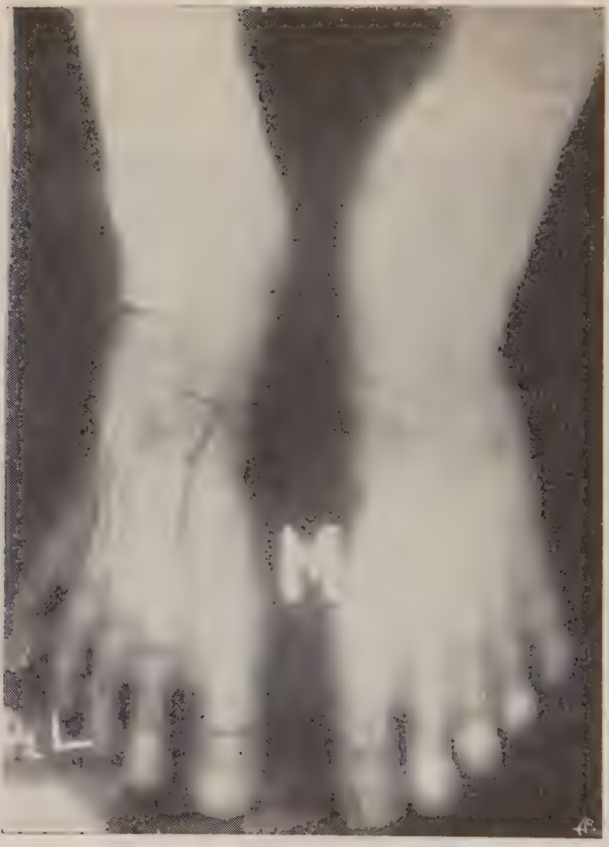


Fig 9 — Idem in the feet

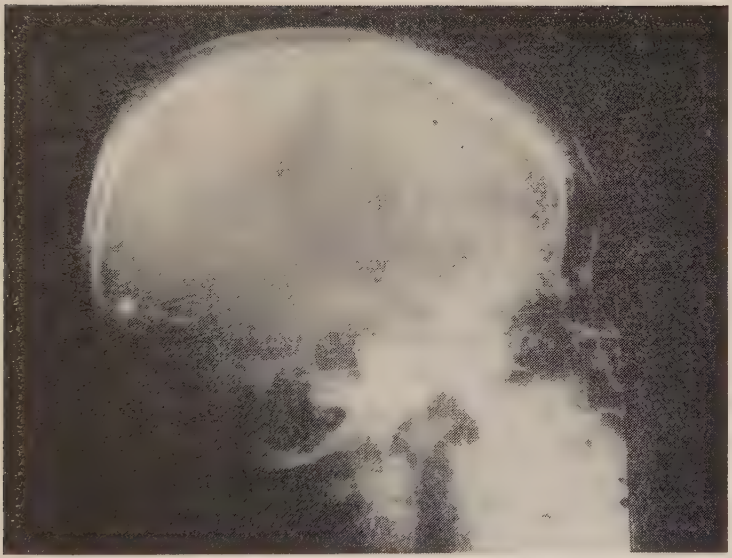


Fig. 10 – Thickening of the cortex of the skull in a case of Hb E-thalassaemia disease in an adult

Sunda Islands (Lie-Injo Luan Eng, 1955, 1957 and additional unpublished data). Hb E has also been found in the Philipines (Stransky and Campos, 1957). In Ceylon Hb E is found in 2 to 3,6 % of the Veddas (Graff *et al.*, 1955, Aksoy *et al.*, 1955).

In Calcutta, India, Hb E was found in less than 4 % of the population (Chatterjea, 1957). Hb E has also been found in Turkey (Aksoy, 1957) and in Serawak (mentioned by Lehmann, 1957). A few cases have been reported from other countries. Hb E has not been found in 907 pure Chinese in Thailand by Na-Nakorn (1957) and we have also not found Hb E in 200 pure Chinese residing in Indonesia, but Vella in Singapore found a very low incidence in what was thought to be pure Chinese. McFadzean also found a low incidence of Hb E in Chinese residing in Hongkong (personal communication).

The relation between Hb E and malaria has been studied in Thailand (Na-Nakorn *et al.*, 1956), but scientific approach seemed inadequate and, therefore, the result was not convincing. Studies on induced malaria in volunteer Hb E trait carriers carried out by Chatterjea in India were inconclusive.

Hb E is in South East Asia nearly as important as Hb S is in the Negro population of Africa and the United States. Together with Hb S, they form the most important haemoglobin problems in the world.

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A METHOD FOR THE DETECTION OF FŒTAL HAEMOGLOBIN AT LOW CONCENTRATION IN BLOOD: SELECTED RESULTS OBTAINED IN CONDITIONS INVOLVING SICKLE-CELL AND OTHER ABNORMAL HAEMOGLOBINS

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For many years fœtal haemoglobin (Hb-F) has been distinguished from the adult type (Hb-A) by its greater resistance towards alkali-denaturation (Korber, 1866). These two closely-related proteins differ in many physical properties (for discussion and literature, see White and Beaven, 1954, 1958; Itano, 1957), and some of these form the basis for the estimation of Hb-F. Differences in the fine-structure of the ultraviolet absorption spectrum of the globin moiety (Jope, 1949) has been utilized. Recent techniques of chromatography on resin columns (Prins and Huisman, 1955; Huisman and Prins, 1955) and electrophoresis on agar gel (Robinson, Robson, Harrison and Zuelzer, 1957) also distinguish Hb-F from Hb-A and from other haemoglobin variants. However, the reaction with alkali remains the most widely-applied in practice.

QUANTITATIVE ESTIMATION OF HUMAN HB-F.

Alkali-denaturation methods. — Since its introduction by Brinkman and Jonxis (1935, 1936) the alkali-denaturation rate method

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has been widely used, though not to the same extent as the more recent Singer 1-minute residue method (for references see White and Beaven, 1954; Itano, Bergren and Sturgeon, 1956). The most recent comparison of the two methods has been made by Jonxis and Huisman (1956), who concluded that at high levels of Hb-F Singer 1-minute values are about 10 % low, while the denaturation rate method is inaccurate below 10 % Hb-F.

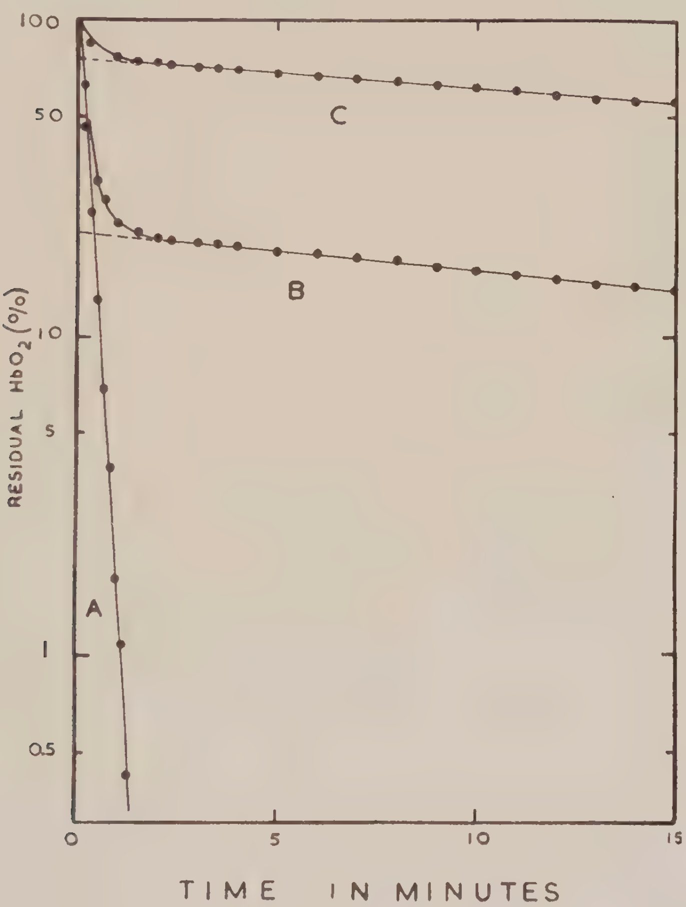

Fig. 1 — Alkali denaturation rate plots of HbO_2 samples: A, normal adult; B, infant, age 4 1/2 months, with sickle-cell/Hb-C disease, 21 % Hb-F; C, cord blood, 76 % Hb-F

The conversion of HbO_2 into alkaline haematin is accompanied by a colour change from red to brown, which can readily be followed spectrophotometrically or colorimetrically at wavelengths appropriate to either HbO_2 or the product. Over a certain pH range, and with controlled alkali-protein ratio, the reaction is kinetically first-order so that a plot of log (unchanged HbO_2) *v* time is linear, with slope proportional to the first-order reaction constant (K_1). The time for half-reaction, *i.e.* 50 % conversion, is also simply related to K_1. Either K_1 or t ½ can thus be used to characterise the reaction. At 20°C and pH 12.8 (N/25 alkali) the values of t ½ for Hb-A and Hb-F are *ca.* 11 and 1,030 seconds respectively, corresponding to K_1 values of 0.063 and 0.0067 $sec.^{-1}$ respectively. It can easily be shown from these figures that the 1-minute reaction time used in the Singer residue method corresponds to *ca.* 99 % conversion of Hb-A and to less than 4 % conversion of Hb-F into alkaline haematin.

When both Hb-A and Hb-F are present, the plot of log (unchanged HbO_2) *v* time is not entirely linear; the initial section of high slope corresponding to the conversion of the Hb-A component, changes smoothly into a final essentially linear section corresponding to the slower reaction of the Hb-F. This linear portion can be extrapolated to zero time to give the proportion of Hb-F in the sample (Fig. 1) with reasonable precision. In practice the method is reliable down to *ca.* 10 % Hb-F; below this level the observations required to establish the linear part of the plot involve measurements of small changes in absorbance over long periods of time and may be subject to instrumental errors, etc.

Singer, Chernoff and Singer (1951) allowed an alkaline solution of HbO_2 of specified concentration and pH to react for exactly one minute at room temperature; the reaction mixture was then neutralized, the denaturation product (alkaline haematin) precipitated and the proportion of unchanged HbO_2 remaining in solution estimated and reported as Hb-F. They found normal adult bloods invariably gave 1-minute residues in the range 0.5-1.7 % and therefore recommended that values below 2.0 % should not be regarded as certain evidence for the presence of Hb-F. This proviso has not always been applied by users of the Singer 1-minute residue method and in consequence many interesting reports of the occurrence of Hb-F below the 5 % level are not entirely reliable. At higher levels, above 10 %

Hb-F, Singer 1-minute residue figures are much more reliable (*v supra*).

Spectrographic methods. — These are based on the observation by Jope (1949) that the tryptophan fine-structure band in the globin absorption band of haemoglobin (Fig. 2) occurs at a shorter wavelength (289.8 mμ) in cord blood (*ca.* 80 % Hb-F) than in adult blood

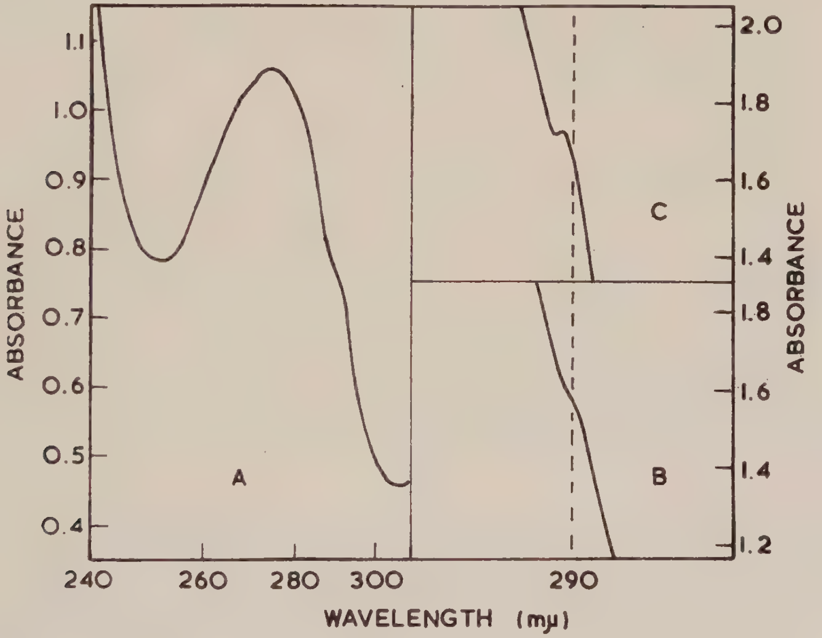

Fig. 2 — Spectroscopic difference between human adult and fœtal haemoglobins: A, absorption spectrum of adult HbCO (globin absorption band) over wavelength range 240 mμ-300 mμ; B, part of globin absorption band in 290 mμ region at high absorbance, showing tryptophan inflection in adult HbCO; C, as for B, but fœtal HbCO at high absorbance, showing resolved tryptophan fine-structure band (100 % Hb-F prepared by controlled alkali treatment of cord blood)

(291.0 mμ). For the location of the fine-structure band, which in Hb-A is only an unresolved inflection (Fig. 2), Jope used a rather specialised, but very convenient method devised by Holiday (1937), in which the photographic plate of a quartz spectrograph is moved by a logarithmic cam to give a relative overall exposure ratio of 1/100.

The resulting spectrogram is particularly well-suited for the detection and location of fine-structure features which do not show up well on conventional plots of absorption spectra (Holiday, 1950). Jope's observation was extended by Beaven, Hoch and Holiday (1951) to the analysis of Hb-F/A mixtures and shown to be accurate to ± 5-10 % over the range 10-90 % Hb-F, but insensitive below the 10 % level. In this form the method has been applied to the estimation of Hb-F in a wide variety of blood samples (Beaven and White, 1953; White and Beaven, 1954). It was used by Liquori (1951) to demonstrate the presence of Hb-F in Cooley's anaemia bloods.

A variant of the method applicable to commercial photoelectric spectrophotometers was devised by Rich (1952), who used the absorbance increment over the wavelength range 288.0-289.0 mμ to characterise the slope of absorption curve in the region of the tryptophan band, after normalisation to a standard absorbance value at 290.0 mμ. The method was used by Rich to estimate Hb-F in Cooley's anaemia bloods. Although it can be carried out on readily available equipment, high experimental precision is required to obtain reliable results, and the errors appear to be most serious with low proportions of Hb-F (Beaven, Ellis and White, unpublished work).

Combined residue and spectrographic method. — In order to extend the sensitivity of the spectrographic method below the 10 % Hb-F level Beaven, Ellis and White (1956) have applied it to alkali-denaturation residues, as obtained in the Singer method, but with a reaction time of ¾ minute and 2.5 % HbO_2 concentration. By combining these two procedures Hb-F may be estimated down to the 1 % level and traces detected to about 0.5 %. In addition a correction for chemical damage to the haemoglobin in the residue may be applied in the form of a protein/haem. ratio, obtained by absorbance measurements at selected wavelengths. This correction was introduced after it was found that Singer 1-minute residues from normal adult blood had abnormal protein/haem. ratios (2 or higher) due to loss of haem. Residues from bloods containing more than 10 % Hb-F have protein/haem. ratios much nearer unity and the correction rapidly becomes smaller as the level of Hb-F rises above this limit. The combined residue-spectrographic method has been found to be of great value in studies of the survival of Hb-F in early infancy, and of its occurrence with other haemoglobin variants in the genetically-determined haemoglobinopathies. Apart from the high levels of Hb-F encountered in Cooley's anaemia (thalassaemia major) such studies require a method suitable for Hb-F levels in the range 1-20 %.

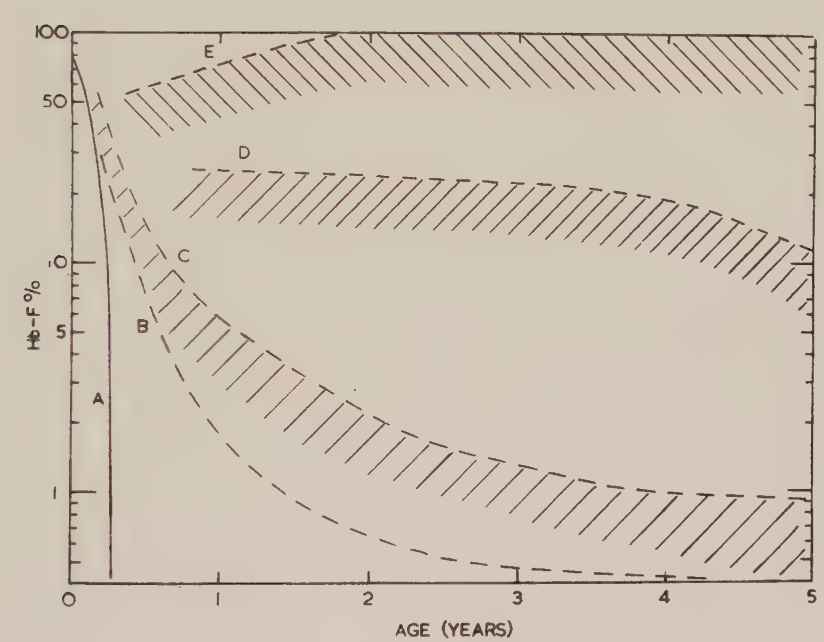

Fig. 3 — Fœtal haemoglobin levels from birth to 5 years: A, curve corresponding to assumption of *linear* disappearance of Hb-F (80 % at birth) with the mean red cell life of 100 days, extended to below 1 % survival; B, average Hb-F levels in a series of child hospital patients with illnesses not involving abnormal haemoglobins; C, approximate upper level of Hb-F in cases of thalassaemia minor and sickle-cell trait; D, approximate upper level of Hb-F in cases of sickle-cell anaemia, heterozygotes for two abnormal haemoglobins, or thalassaemia and an abnormal haemoglobin (i. e. sickle-cell/Hb C disease, sickle-cell/thalasaemia); E, approximate upper level at *ca.* 100 % Hb-F, for cases of thalassaemia major. (The Hb-F values in the range below 1 % are at the useful limit of the combined residue/spectrographic method and should be regarded only as indicating «Hb-F trace less than 1 %». Hb-F values below this limit would be regarded as nil)

PHYSIOLOGICAL OCCURRENCE OF HB-F.

Infancy and childhood. — Beaven, Hoch and Holiday (1951) found no more than 10 % of Hb-F persisting 4 months after birth, and it is difficult to detect any by the ordinary methods in older infants. However, Singer, Chernoff and Singer (1951) and Jonxis and Visser (1956) thought that small percentages might be found for as long as 1 ½-2 years in the blood of healthy infants. Using the combined alkali-denaturation and spectrographic method (Beaven, Ellis and White, 1957) we have not found more than traces (< 1 %) of Hb-F after the first few years of life, and the persistence of higher levels of Hb-F in older children appears to be associated with hereditary haemoglobinopathies or occasionally with other severe blood disorders (Fig. 3). While it is clear that cells containing Hb-F continue to be formed after birth, this process is very limited in degree and finally ceases altogether in health.

Normal adult life. — Despite much work, it is still not clear whether a minute trace of Hb-F may persist in normal adults. Chernoff (1953) has claimed to demonstrate traces immunologically, and Jonxis and Huisman (1956) believe the Singer residue is a mixture containing some Hb-F, and that this is supported by the amino-acid composition (Huisman, Jonxis and Dozy, 1955). Beaven, Ellis and White (1957) have found no spectrographic evidence for Hb-F in residues from normal adult blood, with a limit of sensitivity of 0.4-1 % in the original sample.

PATHOLOGICAL SIGNIFICANCE OF HB-F.

It has frequently been considered that Hb-F may persist after birth in abnormal amounts or reappear in various pathological conditions (see Barcroft, 1934). The evidence is somewhat conflicting, and although there is clear evidence for the post-natal existence of Hb-F in a number of conditions, the mechanism of formation and teleological significance is by no means clear.

Rubowitz (1933) suggested that with residence at high altitudes the low oxygen might stimulate formation of Hb-F. In our own work, we have found no abnormal persistence in children with congenital cyanotic heart disease, in long-continued cyanosis due to drugs, or in the myeloid hyperactivity of myeloid metaplasia. Despite pronounced marrow activity in hypochromic anaemia due to iron-lack or blood loss, Hb-F appears to be absent.

The resemblance of megaloblasts to primitive embryonic erythroblasts and the presence of abnormal macrocytes has suggested the presence of Hb-F in pernicious anaemia (see Larsen, 1951), though tests other than alkali-denaturation did not confirm this (Iversen and Larsen, 1956). By the sensitive combined method, we have found traces ($< 5\%$) of Hb-F in some untreated cases, disappearing slowly with therapy. In some cases of refractory anaemia with hyperplastic marrows small amounts have been detected, although usually absent.

The acquired haemolytic anaemias do not possess Hb-F, and it is usually absent in congenital spherocytic, elliptocytic and atypical forms, though traces occasionally occur (Singer, Chernoff and Singer, 1951; White and Beaven, 1954). Conditions with pronounced extra-medullary haemopoiesis might be expected to form Hb-F, but it is usually absent or present only in small amounts in myelosclerosis and most leukaemias. Occasionally, however, children with leukaemia form large amounts of Hb-F (Beaven and White, 1953).

THE HEREDITARY HAEMOGLOBINOPATHIES.

The hereditary haemoglobinopathies include quite a wide range of haematological disorders in which there is expression of the genes for thalassaemia, sickling or for the formation of other «abnormal haemoglobins». It is in this group that abnormal persistence of Hb-F in post-natal life is particularly found.

Thalassaemia. — In the severely anaemic, homozygous major form of thalassaemia large amounts of Hb-F are of frequent and well-established occurrence (Vecchio, 1946; Liquori, 1951; Rich, 1952). We have found a range of less than 20 to more than 90 % in this condition. The amount of Hb-F is not particularly related to severity (Sturgeon, Itano and Bergren, 1955), and the markedly defective production of Hb-A appears to be part of an inability of the hyperplastic bone marrow to form and deliver mature erythrocytes into the circulation (Sturgeon and Finch, 1957). The persistence of Hb-F in different degrees in young children with major and minor forms of thalassaemia is illustrated in Fig. 4.

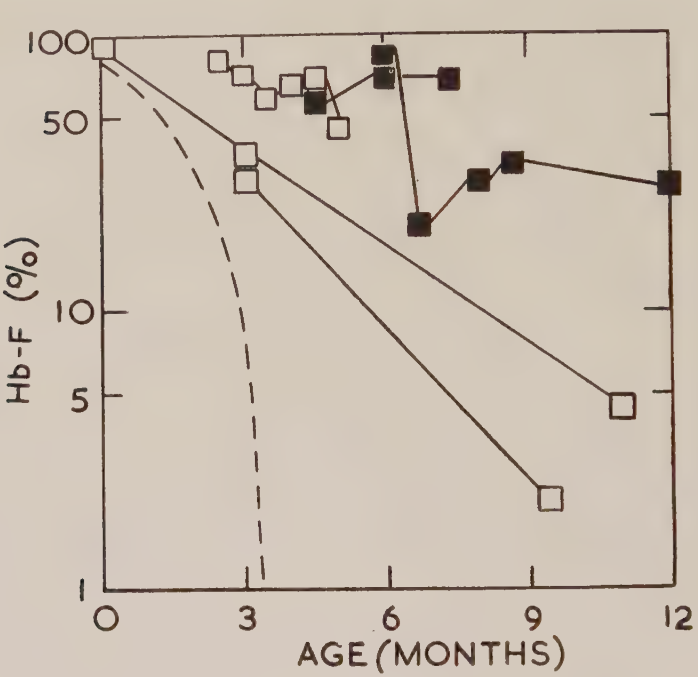

Fig. 4 — Variation of Hb-F levels with time over the period birth to 1 year in infants with thalassaemia minor (□) or thalassaemia major (■). Each set of linked points refers to a single case. The dotted line corresponds to the assumption of linear disappearance of Hb-F contained in red cells with an average life of 100 days

A trace of Hb-F may be found in adults with heterozygous thalassaemia minor (Singer, Chernoff and Singer, 1951) and we have found that about half these individuals have 1-4 % of Hb-F and that this is a useful diagnostic feature. The relationship of Hb-F to Hb-A and the A_2 minor fraction in the various forms of thalassaemia has been discussed by Marinone and Bernasconi (1958).

Thalassaemia is very widespread geographically, occurring widely in the Middle-East and India as well as the Mediterranean area. In the British Isles, rare examples occur (Harvard, Lehmann and Scott, 1958). We have studied five families in which an hereditary and hypochromic anaemia of this type has occurred, and the finding of small quantities of Hb-F in various members has been a valuable aid to diagnosis.

Sickle-cell disease. — The sickling phenomenon is not easily elicited in the blood of young infants, and as the Hb-F declines during the first months of life, Hb-S appears in the cells and these can then be sickled (see Schneider and Haggard, 1955). It is well-known that in homozygous sickle-cell anaemia, although Hb-S forms the bulk of the haemoglobin, up to 20 % of Hb-F may accompany it. The Hb-F may exert some protective function, since it does not enhance the gelation of reduced Hb-S (Singer and Singer, 1953). In the heterozygous sickle-trait condition, the Hb-S is accompanied by an excess of Hb-A, and we have found only traces of Hb-F present in about half the individuals examined.

Other abnormal haemoglobins and mixed syndromes. — Homozygous states for «abnormal» haemoglobins, such as C and E may exhibit small amounts of Hb-F, but particularly notable amounts of up to 20-30 % of the total may occur in the mixed syndromes due to the inheritance of the thalassaemia gene and a second gene for Hb-S, C or E. In these states, the formation of Hb-A appears suppressed to varying degrees, the abnormal haemoglobin predominates, but a varying proportion of Hb-F co-exists (see Itano, Bergren and Sturgeon, 1956; MacIver, Went and Cruickshank, 1958).

Though the question has been raised whether the Hb-F found in pathological blood is identical with the normal fœtal pigment (Perosa and Bini, 1954), most workers agree that this is so (Itano, 1957). Our own use of the simultaneous spectroscopic and alkali-denaturation criteria support this conclusion.

Genetic aspects of Hb-F. — The genetic pattern for the production of Hb-F and its normal replacement by Hb-A is obscure. The persistence of Hb-F is of great interest in the many instances of operation of genes for the Mendelian-dominant abnormal haemoglobins, most marked where homozygous, much less so where heterozygous with the gene for Hb-A production. The marked influence of the gene for thalassaemia in continued Hb-F production is also very evident when heterozygous with a further gene for an «abnormal haemoglobin». The possibility of double-heterozygosity may be related to the great variability of Hb-F and Hb-A in such conditions and calls for careful familial as well as biochemical studies (Zuelzer, 1957). The possibility that Hb-F itself may persist into adult life as a genetic entity without associated haematological abnormality has been raised by Jacob and Raper (1958).

SUMMARY

Fœtal haemoglobin can be detected in the presence of adult haemoglobin by its greater resistance towards alkali-denaturation and by its ultraviolet spectrographic characteristics. However, the distinction is most accurate when the proportion of Hb-F is high, and when it is less than 10 % of the total both methods lack sensitivity.

Small proportions of Hb-F frequently occur in the milder, heterozygous forms of thalassaemia or conditions where an abnormal haemoglobin is found, as well as in various anaemic states which are not of an overtly genetic character. In such cases, the use of a sensitive combined method of partial alkali-denaturation and ultraviolet spectrographic examination of the residue enables Hb-F to be determined to a limit of 0.4-1 %.

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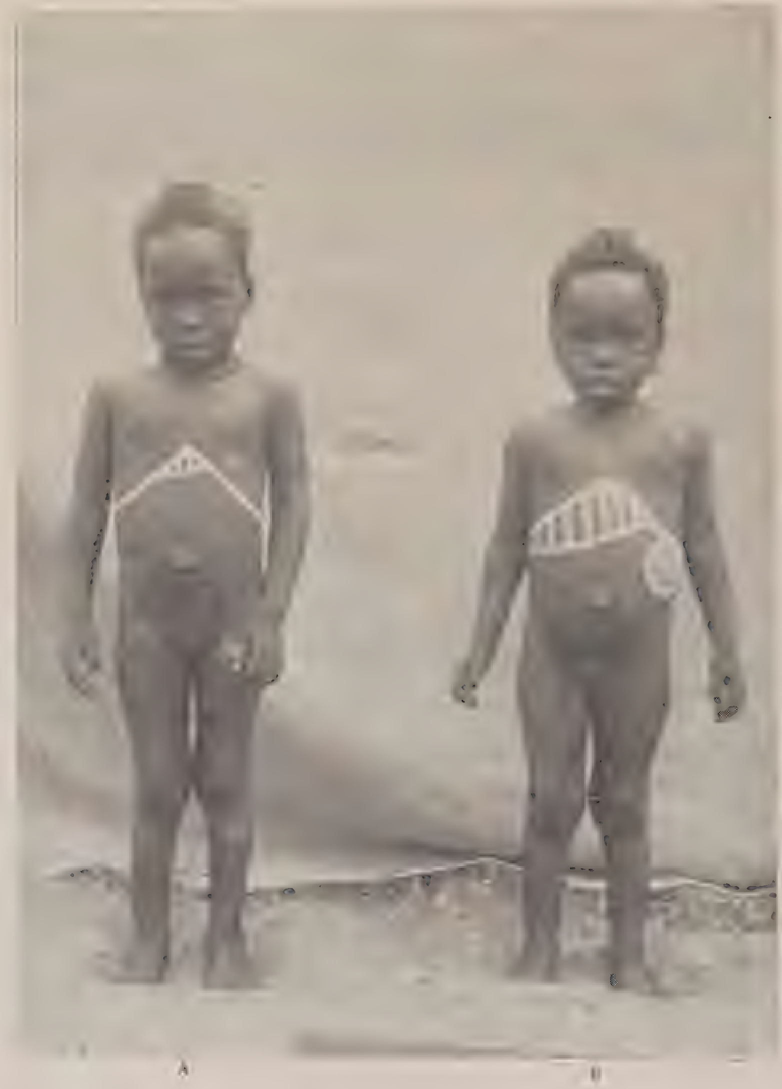

Homozygous twins aged 3 1/2 years. *A*, protected from malaria from birth. *B*, unprotected from malaria

contrast to populations living in temperate climates an increase in gamma globulin in Africans does not necessarily indicate a pathological condition. The gamma globulin comprises antibodies, and a highly immunised group would necessarily show a higher gamma globulin level. This in Africans tends to cause a rise in zinc sulphate turbidity levels, but not necessarily in thymol turbidity values. The first simply indicates a high gama globulin, the second is more specifically related to a rise of gama globulin in association with liver damage. In 12 instances the thymol turbidity was examined and it was found to be above the normal value in 7. In these 7 instances serum electrophoresis in 5 showed nothing but the high gamma globulin, but in 2 the pattern was of the type characteristic of advanced cirrhosis. The results of the liver function tests are set out in Table 1 and have been arranged in sequence of age.

Despite the incompleteness of these data, it is clear that the majority of the subjects showed evidence of liver cell damage with, in certain instances, bile retention.

There is some indication that the damage progresses with age and this is born out by findings made in 3 patients who were observed over periods of 15 to 22 months. No. 12, a boy of 4 years when first seen, was re-examined after 22 months: during this period the size of his liver had increased from 2 fingers breadth to 5 fingers breadth below the right costal margin, while the spleen, formerly palpable to one finger's breadth, had become impalpable. The concentration of serum albumin fell from 4.4 g to 4.0 g per 100 ml, while that of the serum globulin rose from 2.8 g to 3.1 g. The serum bilirubin increased sligthly from 0.6 mg to 0.8 mg/100 ml and the direct Van den Bergh reaction became weakly positive. The serum pseudocholinesterase level fell from 62 units (rather low for a child but perhaps still just within normal limits) to the definitely pathological value of 48 units, while both thymol and zinc sulphate turbidities rose from 2.5 to 8 units and from 10 to 32 units respectively. In the case of no. 25, a male child aged 5 years when first seen, the liver size increased from 5 to 6 fingers breadth and the spleen, initially palpable to 5 fingers breadth, became impalpable. In no. 3, a boy of 9 years, the degree of hepatomegaly remained the same at 3 fingers breadth, but the spleen at first palpable to one finger's breadth had become impalpable after a period of 15 months.

TABLE 1

Serial number	4	21	2	5	8	12	25	16	15	22	13	27	23	18	17	7	24	3	20	11	
Age	2 1/2	2 1/2	3 1/2	3 1/2	3 1/2	4	5	6	7	7	9	9	13	14	16	18	19	26	31	35	Normal values in Europe
Sex	M	M	F	F	M	M	M	M	M	F	M	F	M	M	F	M	M	M	M	M	
Albumin (g/100 ml)	—	4.8	—	4.2	4.6	4.4	—	4.7	3.4	4.7	5.0	—	—	—	—	—	—	—	4.1	2.7	4.0–6.7
Globulin (g/100 ml)	—	1.6	—	3.0	4.6	2.8	—	3.4	4.0	3.4	3.6	—	—	—	—	—	—	—	3.5	5.4	1.2–2.9
Bilirubin (mg/100 ml)	0.7	0.7	3.7	—	2.0	0.6	—	2.4	1.5	1.1	2.1	—	—	—	2.4	—	—	0.8	3.5	0.5	Up to 0.8 mg 100 ml
Direct Van Den Bergh	0	0	++	—	++	0	—	+	+	0	+	—	—	—	+	—	—	+	++	0	
Pseudocholinesterase (units)	57	62	72	—	—	62	—	100	79	49	63	—	—	—	64	—	—	24	34	24	55–110 higher in children
Thymol turbidity (units)	1	5	8	—	—	2.5	—	2	1.5	5	2	—	—	18	—	—	—	5.5	4	7	Up to 2 units
Zinc sulphate turbidity (units)	7	—	21	—	—	10	—	20	14	24	12	—	—	—	—	—	—	7	—	30	Up to 4 units
Alkaline phosphatase (K. A. units)	18	—	—	—	18.5	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3–18 units. Higher in children

The results of this casual investigation stimulated us to undertake a formal investigation of liver function in sickle-cell anaemia and to try to relate the anomalies found to progressive change in the structure of the organ.

LIVER FUNCTION IN A SECOND GROUP OF CASES OF SICKLE-CELL ANAEMIA

Following the random study of cases of this disease among the Lagos African population, a more formal investigation was carried out in which 16 patients, whose ages ranged from 1 ½ to 30 years, were admitted to a special ward. The same range of biochemical investigations were performed and the results are tabulated in Table 2.

Though the haematological findings in these patients indicated the phenotype SS, it was possible to prove the genotype by examination of blood specimens from both parents in 5 instances only. Confirmation of the genotype as SS was thus obtained in each case, the relevant serial numbers being 7, 16, 18, 19 and 25.

The following points may be made:

a) Bile retention with a positive direct Van den Bergh reaction was evident in 6 out of 8 patients above the age of 6 years. In the younger children the excess of bilirubin found in 3 out of 7 subjects was associated with a negative diazo reaction and was evidently of the prehepatic type.

b) Serum globulin levels tended to increase with age, while among patients whose serum albumin concentration was less than 4 g per 100 ml 3 were noted out of 5 patients aged 10 years and over but only one among the 11 patients who were younger than this.

c) Pseudocholinesterase levels, which show a range in health of 55 to 110 units but give higher values in children, appeared to be pathologically low in the 3 youngest children, aged 1 ½, 1 ½ and 4 years, and were within the low normal range in all but 2 of the remainder. A significant increase in excess of 10 units was observed to follow rest in hospital in 9 instances.

d) Thymol turbidity tests gave a normal value in only one patient, aged 6 ½, were borderline in 5, aged 4, 4, 6, 9 and 11 years respectively, and clearly abnormal in the remaining 10. Above the age of 10 years one patient only out of 5 showed a borderline result and in the remainder the values were abnormal. Below this age there were one normal value, 4 borderline values and 6 abnormal results.

e) Zinc sulphate turbidity tests, however, gave high results in every case. Very high values in excess of 20 units were obtained in 3 out of 5 patients above the age of 10 and in 3 out of 11 younger patients.

f) Serum alkaline phosphatase levels were not uniform, but were raised in excess of 30 K-A units on occasion in 3 patients, whose ages were 6 ½, 9 and 11 years. The biochemical picture is again one of liver cell damage and of bile retention which progress as age increases.

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HISTOLOGICAL CHANGES IN THE LIVER IN SICKLE-CELL ANAEMIA

J. H. WALTERS

Punch biopsy offered an explanation of the biochemical observations. The changes observed are illustrated by photomicrographs and may be summarised as follows:

a) Focal necrosis of liver cells was negligible in the youngest patients, was more evident in 2 older children of 6 ½ and 11 years, and was gross in the only adult aged 30 years.

b) In all specimens sinusoidal dilatation was evident (compared with control specimens obtained, fixed and stained in an identical manner) and in each patient (but in no control material) the sinusoids contained a delicate fibrinoid reticulum which appeared to be in continuation with the stromal fibres of the perisinusoidal membrane.

c) There was evidence that this reticulum is progressively incorporated beneath the endothelium into the perisinusoidal stroma, which becomes thickened thereby. In the adult patient this thickening was so gross as to constitute a broad lamellar deposit of loose fibrous tissue separating attenuated plates of liver cells from patent though narrowed sinusoids.

d) Sometimes sinusoidal blockage by clumps of sickled erythrocytes is evident: this may lead to focal liver cell necrosis with subsequent scarring.

e) Silver impregnation methods show with increasing age a progressive thickening of the longitudinal stromal fibres which amounts in adult life to a characteristic fine cirrhosis with dense localised plaques of fibrosis which result from superadded focal necrosis.

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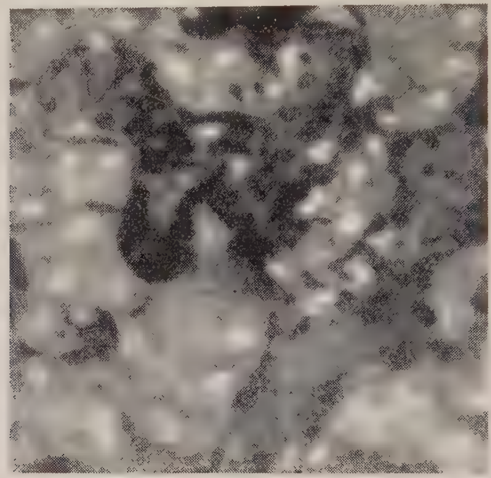

Fig. 1 — Liver biopsy specimen, alcohol fixed, stained with haematoxylin and eosin, to show fibrinoid reticulum within sinusoids

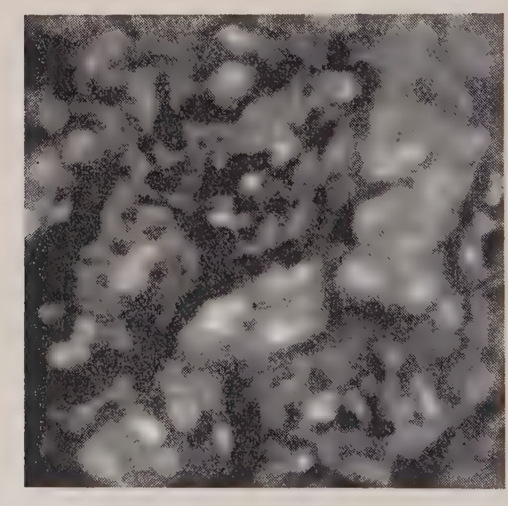

Fig. 2 — Liver autopsy specimen, formol fixed, showing intact sickled erythrocytes enmeshed in reticulum

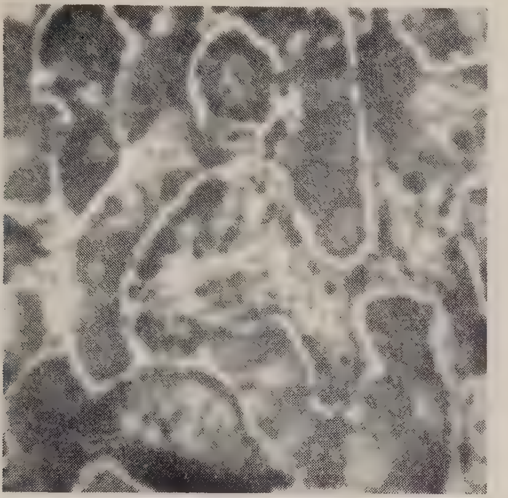

Fig. 3 — Liver biopsy specimen, alcohol fixed, showing reticulum impregnated with silver

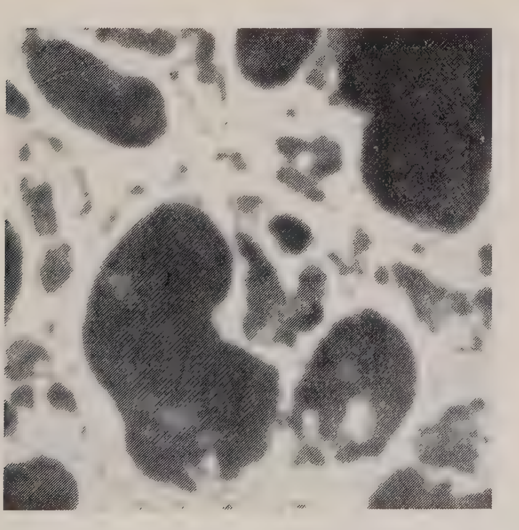

Fig. 4 — Liver autopsy specimen, showing gross thickening of peri-sinusoidal membrane due to incorporation of fibrinoid material onto it

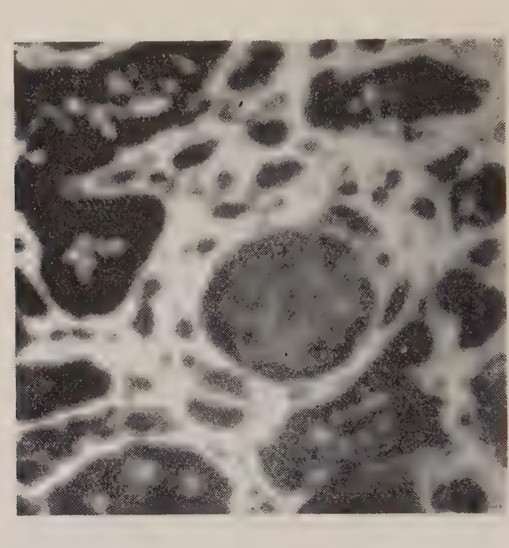

Fig. 5 — Liver autopsy specimen, showing lamellar thickening of wall of sinusoid seen in cross section

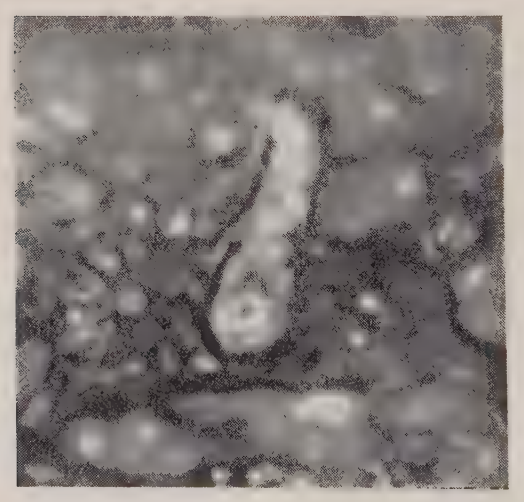

Fig. 6 — Liver autopsy specimen, showing endothelial wall of a sinusoid separated from liver cells by a broad zone of fibro-cellular tissue

CONCLUSIONS

The conclusions drawn from these observations are that the fibrinoid reticulum observed in all the specimens is laid down in life and is associated with hyperfibrinogenaemia and increased prothrombin activity. Its presence must impair the circulation within the hepatic sinusoids and therefore facilitate sinusoidal plugging. Incorporation of this fibrinoid material leads to a progressive thickening of the perisinusoidal membrane which prejudices the diffusion of gases and electrolytes across it. Increasing fibrosis aided by gradual attrition of liver cells and by focal necrosis due to acute sinusoidal obstruction culminates in a cirrhosis of characteristic pattern if adult life be attained.

ERYTHROCYTE SURVIVAL IN SICKLE-CELL ANAEMIA

S. M. LEWIS

Erythrocyte survival studies have been carried out by the ^{51}Cr method on three patients with sickle-cell anaemia. Special precautions were taken to ensure that the blood was well oxygenated throughout the *in vitro* labelling process, after which the labelled erythrocytes were re-introduced into the patients' circulations.

In all patients a double cell population was demonstrated. One component was eliminated from the circulation at an extremely rapid rate, whereas the second component was eliminated at a slower rate, although in all cases this was more rapid than normal. The pattern of elimination during the first phase was curvilinear; that of the second component was shown on arithmetic graph paper to be linear.

	Population A		Population B		Effective mean cell life
	% cells	M. C. L.	% cells	M. C. L.	
Case 1.	35	4 days	65	66 days	10 days
Case 2.	30	5 »	70	80 »	14 »
Case 3.	65	6 »	35	38 »	8 »

Body surface counting by a sensitive scintillation counter showed that there was a significant accummulation of radio-activity in the liver, but no excess activity in the spleen. The process of liver accummulation was progressive throughout the life span of the circulating labelled erythrocytes. This suggests that the liver is the site of cell sequestration and destruction in sickle-cell anaemia.

It has been the only disease amongst a large series of haemolytic anaemias, congenital and acquired, in which this pattern has been observed.

3RD SESSION: *Saturday, September 6th — 11:00 to 12:30 a.m. Room A*

The meeting was held under the chairmanship of Dr. M. Payet.

The Rapporteurs were Dr. A. W. Woodruff and Dr. Carlos P. Trincão and the Secretary was Dr. Egídio R. R. Gouveia.

The following papers were presented and discussed.

ANAEMIAS OF IMPORTANCE TO THE PUBLIC HEALTH IN THE TROPICS

A. W. WOODRUFF, M.D., F.R.C.P., D.T.M. & H.

Wellcome Professor of Clinical Tropical Medicine, London
School of Hygiene & Tropical Medicine; Physician, Hospital
for Tropical Diseases, London

Anaemias have for several generations attracted the attention of physicians in the tropics, who have not been slow to realise that they are common causes of morbidity and even mortality there. To-day the principal questions relating to them are firstly how important are they as a public health problem? Secondly what are their causes and thirdly what is to be done about them? My personal experience in this field is based on work carried out during the last 16 ½ years in India, Burma, West Africa and Mauritius and an attempt will be made to place before you the elements of this experience.

THE IMPORTANCE OF ANAEMIA TO THE PUBLIC HEALTH

The prevalence of anaemia and its importance to the public health are difficult to assess on a community basis. Most hospital statistics give little information on these points because the population served by a particular hospital is so seldom known and only infrequently can it be ascertained that patients in the region suffering

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from anaemia do not seek treatment elsewhere. But if a disease is common in a hospital, it is safe to deduce that it constitutes a considerable problem in the population served by that hospital. Even so the collection of accurate data concerning the prevalence of the different types of anaemia in the tropics is one of the greatest present needs in this field. Some personally collected figures on their prevalence are relevant here for these underline their importance. The relatively compact yet densely populated island of Mauritius is served by a network of small hospitals which makes it one of the few places where comprehensive data of this kind can be collected and the value of the data is enhanced by the fact that malaria, considered by many to be an important aetiological factor in connection with anaemia, has recently been eradicated from the island, so that one of the important variables in considering aetiology has been removed. The relative importance of anaemia as a cause of morbidity in Mauritius can be assessed by comparing numbers of patients treated for it in hospitals there with the numbers of those treated for other diseases. Thus in 1948 only accidents, diseases of the skin and malaria were responsible for more admissions yet in 1953, by which time malaria had been completely eradicated, only admissions for accidents exceeded those for anaemia (Table 1).

TABLE 1 — *Admissions to hospitals in Mauritius in 1948, just before malaria eradication was commenced and in 1953 after the completely successful conclusion of the malaria eradication scheme*

	Numbers of cases treated in hospitals	
	1948	1953
Accidents, poisoning and violence	3,607	4,025
Diseases of the skin and cellular tissues	2,674	1,303
Malaria	1,976	0
Anaemia	1,592	2,934
Bronchitis	752	921
Influenza	618	857
Ancylostomiasis	537	431
Tuberculosis	525	555
Gastroenteritis and colitis	419	408
Syphilis	278	153

According to census figures the population of Mauritius on 31st December, 1954 was just over half a million. The figures in Table 1 reflect only a proportion of the persons on the island suffering from anaemia, many do not go to a medical practitioner or are not admitted to hospital; it may well be that there are twice or three times as many with well marked anaemia as are shown in the table but even as they stand the figures indicate that 1 person in 250 there is anaemic and that the absolute number of those suffering from anaemia is very considerable.

The relative degrees of severity of anaemias encountered in Mauritius are indicated in Table 2 — kindly made available to me by Dr. N. Chung Hin, Government Pathologist, Mauritius. During the three months commencing 1st July, 1955 there were referred to the Central Laboratory and its two branches in Mauritius 2,051 persons who were found to have less than 14.0 g haemoglobin per 100 ml blood. Of 1,200 Indians 26 % had 6.4-9.1 g haemoglobin per 100 ml blood and 34 % had less than 6.4 g per 100 ml. The Creole community appeared to be less severely affected for of 567 who were referred 11.5 % had 6.4-9.1 g per 100 ml and 11.8 % had less than 6.4 g per 100 ml.

TABLE 2 — *Severity of anaemias encountered in different racial groups in Mauritius, July-September, 1955*

Total number examined in each group			Hb. 9.2-11.9 g	Hb. 6.4-9.1 g	Hb. 3.6-6.3 g	Hb. 1.3-3.5 g
Indian	1,266	Number of cases found	211	325	308	128
		% of cases in group	16.7	25.7	24.3	10.1
Creole	567	Number of cases found	106	65	55	12
		% of cases in group	18.7	11.5	9.7	2.1
European	178	Number of cases found	24	—	—	—
		% of cases in group	13.5	—	—	—
Chinese	40	Number of cases found	6	4	3	5
		% of cases in group	15	10	7.5	7.5

In the Gambia, Woodruff and Schofield (1957) carried out a survey of haemoglobin values and found that among males aged

17-45 years the mean value was 11.76 g per 100 ml blood, among non-pregnant women of similar age it was 10.12 g, among pregnant women 9.09 g per 100 ml blood and that of 288 persons in the survey 31 or 10.7 % had a haemoglobin value of less than 8.0 g per 100 ml. Means which most workers would agree are far from optimal.

From India there has now for upwards of 30 years been a steady stream of reports on the prevalence of anaemia, particularly in relationship to pregnancy and though I do not propose to refer to this literature now there is one piece of work of particular importance concerning the prevalence of anaemia and that is the study of Hynes, Ishaq and Morris (1945), who found that of 600 recruits to the Indian Army only 195 had a haemoglobin value of 51 % or more. In other words, two thirds of these recruits were suffering from moderate or severe anaemia.

Woodruff (1955*a*) studied anaemia in India on behalf of the World Health Organisation and in each centre visited, was struck by the amount of anaemia there was. At Gwalior, Prof. N. T. Benawri stated (personal communication) that one in 50 school children had a haemoglobin value of 10.0 g % or less. At Indore in Central India, Mukerji and Sepaha (1952) found anaemia to be one of the commonest ailments encountered in the King Edward VII Hospital. They reported that of 1,100 patients admitted to the medical wards of the hospital a little over a quarter had a haemoglobin value of between 8.6 g and 11.5 g %, 2.5 % had values between 5.7 g and 8.6 g %, 3 % had values between 2.9 g and 5.7 g %, and in 1.8 % the value was below 2.9 g % haemoglobin. Dr. P. S. Vasavda, Director of the newly established Department of Haematology at the Medical College Indore, found (personal communication) that between September 1953 and July 1955, 9,710 patients were admitted to the medical wards of the hospital there and that of these, 738 were referred to the Department for anaemia. Of these 524, or 5.4 %, of all admissions to the medical wards had a haemoglobin value of less than 10 g per 100 ml blood, 103 had less than 4.5 g % and an additional 164 a value between 4.5 g and below 7.5 g %.

At Indore, Prof. I. Bhonsale (personal communication) stated that 25 % of women admitted to the Department of Obstetrics and

Gynaecology are suffering from an anaemia with less than 10.8 g % haemoglobin.

Working from the Poonamallee Health Clinic near Madras, George had carried out a survey of the haemoglobin values among infants and pre-school children and found that 83 out of 340 of these, i.e. 24.4 %, had a haemoglobin value of less than 8 g per 100 ml, that only 38 % of them had a value of more than 10 g % and as few as 5.7 % had a value of over 12 g %.

TABLE 3 — *Haemoglobin values in 340 pre-school children living near Madras, 1955*

Age	Number of cases				
	Less than 6 g %	6-8 g %	8-10 g %	10-12 g %	Above 12 g %
Less than 1 year	6	17	19	18	2
1-2 years	9	9	31	15	2
2-3 years	5	11	23	27	6
3-5 years	11	15	53	51	10

In the Ramanagaram District of Mysore State, Krishmaswamy Rao and Sundar Rao have carried out a nutritional and haematological survey of 3,964 persons. The mean haematological values are found in Table 4, from which it will be seen that the mean haemoglobin at all ages is very much less than optimal and that only in males of more than 16 years does it exceed 11.6 g per 100 ml blood.

TABLE 4 — *Haematological values in 3,964 persons in rural Musore*

	Aged 0-6 years		Aged 7-12 years		Aged 13-15 years		Aged 16 years and above	
	Average Hb (g %)	Number	Average Hb (g %)	Number	Average Hb. (g %)	Number	Average Hb. (g %)	Number
Male	10.0	301	10.5	642	10.6	191	11.3	899
Female	9.5	218	10.4	448	10.1	97	9.2	898

In Nagpur in September, 1955, during a personal visit, Prof. J. N. Berry kindly arranged for haemoglobin estimations to be made on 40 male and 40 female patients at that time in the medical wards there. It was found that half the males and all but 7 of the female patients had haemoglobin values of less than 10.15 g per 100 ml and that 13 of the 80 had values below 4.4 g %.

TABLE 5 — *Haemoglobin values in 40 male and 40 female hospital patients (Nagpur, 1955)*

Haemoglobin	Numbers of patients	
	Males	Females
Less than 4.4 g %	6	7
4.4-7.24 g %	3	8
7.25-10.15 g %	11	18
More than 10.15 g %	20	7

THE IMPORTANCE TO THE PUBLIC HEALTH OF ANAEMIA IN PREGNANCY.

The enormous literature on this subject which has been built up during the last three decades is evidence of its frequency and several workers have drawn attention to the risks to life both of mother and infant, resulting from it. It is here proposed to touch only on three pieces of personally known work. Raj (1954) in Nagpur found anaemia to be the commonest single cause of maternal mortality in this region. Forty per cent of the total of 117 maternal deaths in the last five years at the Daga Memorial Hospital, Nagpur, were due to severe anaemias of pregnancy.

At the Medical College Hospital, Nagpur, figures made available by the kindness of the Dean, Dr. G. L. Sharma, indicate that during 1954 there were 1,151 deliveries in the Department of Midwifery and that 178 of the mothers concerned had a haemoglobin value of less than 8 g %; in 39 cases the value was less than 3 g % and in 56 it was between 3 g and 5 g %.

At the Wadhia Maternity Hospital, Bombay, Dr. Radhakrishnan kindly gave me haemoglobin values estimated on the last 1,463 pregnant women who had attended the antenatal clinic there. Their mean haemoglobin value was 8.93 g %, a very low figure for an unselected series of pregnant women. In 33 cases the value was below 5.80 g %, in 8 it was below 4.35 g % and in only 37 cases was it greater than 11.5 g %.

ANAEMIA AND MALNUTRITION IN CHILDHOOD.

There is now a large literature concerning protein malnutrition or kwashiorkor throughout the tropics and it is generally agreed and has been emphasised by Brock and Autret (1952) and by Trowell, Davies and Dean (1952) that the disease is associated with mild or moderate anaemia when uncomplicated by secondary parasitic infection. In India, studies of the prevalence of anaemia in childhood have been relatively few, but important work in this field has been carried out at the Nutrition Research Institute, Coonoor. The region around Coonoor has been found by Someswara Rao *et al.* (1954) to be one «which for all practical purposes is free from hookworm infestation and malaria», and this makes the finding of anaemia there of especial importance in considering the role played by nutritional factors. Secondly it has been found that haematological values for average Indians will not in any way be different from those of persons living in Western Europe and other temperate climates (Indian Council for Medical Research, Special Report Series, No. 26. 953). Thirdly it has been shown by Ramalingaswami and Venkatachalam (1950) that there is no appreciable difference between the mean haematological values of adults living in the Coonoor district as compared with those living in other regions of India. Nevertheless, in the same district a survey of children from poor and lower middle class families revealed that their haematological values were strikingly lower than those of British children and some other Indian children (Someswara Rao, Taskar and Ramanathan, 1954). From the fourth year of life onwards children in the Coonoor series exhibited haemoglobin values of 0.5 g to 2 g per cent lower than those found in British children in 1943 and reported in the Medical Research Council, Special Report series

No. 252. Work also carried out at Coonoor indicates that protein malnutrition similar to that described in Africa is encountered there and is associated with anaemia (Nutrition Research Laboratories, Coonoor, Report for the Year 1949-50).

CONCLUSIONS REGARDING IMPORTANCE OF ANAEMIA IN THE TROPICS.

From this information it is clear that anaemia of various kinds constitutes a common and important problem in India, Africa and Mauritius and what applies there may be extended to most of the remaining tropics. Much maternal mortality appears to be attributable to it and at all ages of life and particularly during pregnancy, it causes much morbidity. It is impossible to obtain a precise assessment of the incidence of the disease from hospital statistics; the population served by the hospital is seldom known with accuracy and many factors including the local influence of indigenous systems of medicine and confidence in the hospital concerned constitute sources of errors in such assessments. Nevertheless, if a disease is commonly found in hospitals in a region it is invariably more common in the general population. Moreover, hospital figures are supported by the evidence provided by surveys not only in Africa and in India but indeed, throughout the tropics.

CAUSES OF THE COMMON ANAEMIAS

IRON DEFICIENCY ANAEMIA.

If one excludes the genetically determined anaemias the commonest cause of those remaining is by general consensus of opinion iron deficiency. Studies of the relative frequency of the different forms of anaemias encountered in hospitals are few but the almost universal experience of physicians in the tropics is that the majority of their patients with anaemia are of this type. Augmenting these impressions there are now the results of carefully conducted studies of groups of persons, one such is that of White *et al.* (1957) who reported dietary surveys from an area in Peru indicating a mean iron intake there of 12 mg per person per day and this was associated with the widespread ocurrence of iron deficiency anaemia, probably aggravated by hookworms. To a group of 50 schoolgirls they therefore administered 50 mg ferrous sulphate daily for 14 weeks and gave a placebo to a similar group of control. Haemoglobin values in the experimental group improved from a mean of 3.39 per 100 ml to 10.21 per 100 ml in 6 weeks and the value remained virtually unchanged after 14 weeks treatment. In the control group no statistically significant change in haemoglobin values occurred. In the experimental group the haemoglobin values had not returned to their initial levels 8 months after cessation of the treatment and had fallen most, in those who before treatment were most severely anaemic — a result which might be expected if the degree of anaemia had been related to the hookworm load, for no anthelminthic treatment was given. A further study of two similar groups showed that daily supplements of 15 mg and of 30 mg of ferrous sulphate were as effective in improving the haematological picture as was the supplement of 50 mg and from this it was concluded that a daily iron intake of approximately 25 mg was required by these hookworm infected children. This amount is considered to be not far above that which would be obtained from food enrichment and dietary guidance.

Probably the most important problem concerning iron deficiency anaemia is that relating to its cause for upon this depends the measures to be taken in its control. The main possibilities are:

1) An inadequate dietary iron content.
2) Excessive loss of iron from the body either as the result of helminthic infection or as Foy *et al.* (1955) have suggested through the loss of iron in the sweat.
3) Bacterial or other non-helminthic infections may be in part responsible, for although the anaemia they cause is usually normocytic and normochromic, it may sometimes be microcytic and hypochromic.

INADEQUATE DIETARY IRON CONTENT.

Regarding the first of these possibilities, and bearing in mind that maybe only a third or less of the dietary iron is absorbed from the alimentary tract, it is generally considered that a daily intake of 15 mg of iron is adequate for basic needs including those of pregnant women and growing children (Whitby and Britton, 1950). Certainly when the dietary intake falls much below the 15 mg level anaemia may become common in tropical populations as has been shown by White *et al.* (1957) — probably particularly if ancylostome infection is common in the population. It appears probable that 15 mg iron is present in the diets of the majority but not all tropical people. Thus Mitra (1953) found 6.5 % of 843 Indian families to have a daily iron intake per person of less than this amount. Radhakrishna Rao (1954) found the iron intake in 37 out of 185 persons in the Humgaon area of Bombay state to be 16.5 mg per day. It is clear, therefore, that considerable numbers of tropical residents have an inadequate or barely adequate dietary iron content and that they are therefore in a poor position to withstand even the small losses of iron which might be caused by light helminthic infections. It seems probable, therefore, that some iron deficiency anaemias in the tropics may be truly dietary in origin. It must, moreover, be remembered that estimates of the amount of dietary iron absorbed from the intestinal tract have been as low as 5 % (Foy and Kondi, 1956).

EXCESSIVE LOSS OF IRON FROM THE BODY.

Iron lost in the sweat. — Attention has latterly been directed towards the loss of iron which may occur in sweat, particularly in cell rich sweat and in this field the work of Foy and his colleagues has been prominent. On the other hand, Moore (1950), using techniques employing radio-active isotopes, has found that even under conditions of maximal sweating only approximately 0.5 mg iron can be lost per day in the sweat. This is a small loss but if, as seems possible in certain regions, less than 5 mg iron are being absorbed from the intestine daily, the amounts of this order might prove important in the causation of anaemias. Foy *et al.* (1955) have emphasised that cells rubbed from the body surface and present in cell rich sweat constitute a considerable source of iron loss.

Iron losses caused by helminthic infections. — 30 years ago ancylostome infections were considered to be the cause of the majority of anaemias occurring in the tropics but gradually the climate of opinion changed and latterly there has been a tendency to consider ancylostomiasis unimportant as a cause of iron loss and anaemia and this tendency has been fostered by the demonstration that iron can be lost in the sweat. The doubt which exists in the minds of many has been reflected by Meiklejohn and Passmore (1958), who have suggested that the time is ripe for a properly financed research project to examine this question. The evidence, however, in favour of ancylostomiasis commonly causing iron losses with consequent anaemia is very strong and accumulated experience, personal and otherwise, points towards such a conclusion. Thus among 27 patients with anaemia studied in Mauritius, 1955 (Woodruff, 1955*b*), 24 or 89 % were found to be infected with *Ancylostoma duodenale*. This compares with an incidence of ancylostomiasis of 35 % among general hospital patients in Mauritius. In other words, ancylostomiasis occurs more than 2 ½ times as commonly among patients with anaemia in Mauritius than among general hospital patients and it may further be presumed that there are more patients with ancylostomiasis among the hospital patients than among the remainder of the population.

The World Health Organisation has, at the invitation of the Government of Mauritius, set up a team under Dr. Gordon Stott for investigating anaemia there. In an experiment which he has carried out, patients have been allocated at random to one of four groups. To those in the first, 400 mg ferrous sulphate have been administered daily; to those in the second, this amount of ferrous sulphate plus 5-10 mg folic acid; to those in the third, this amount of folic acid daily only; and to those in the fourth, a placebo was administered. To each group 60 patients were allocated and it was found that 70 % of those receiving ferrous sulphate only; 65 % of those receiving ferrous sulphate and folic acid; 10 % of those receiving folic acid only and 3 % of those receiving the placebo responded within 30 days with a rise of 2 g or more haemoglobin per 100 ml blood. The findings are consistent with the anaemia being the result of a high incidence of ancylostomiasis and the experiment has further underlined the high prevalence of anaemia of iron deficiency type in Mauritius.

The important results of the application of methods designed to study groups of patients have indicated that ancylostomiasis is a common cause of iron deficiency anaemia in the tropics and this conclusion is underlined further by the results of studies using isotopes to estimate blood loss. Thus Tasker (1958) has found in *Necator americanus* infection that the blood loss is remarkably constant at about 0.1 ml blood per worm per day, a loss which is reduced to a fraction after administration of a vermifuge.

NON-HELMINTHIC INFECTIONS AS CAUSES OF IRON DEFICIENCY ANAEMIA.

Malaria was long considered to be an important cause of microcytic hypochromic anaemia in the tropics, but Garnham (1949) in East Africa found the diseases there to be of little importance in the early years of the African's life, to cause only slight fever, loss in weight, a lowering of the erythrocyte count by only 13 % in the first attack and a return to normality by the fifth month. Others, however, including Wilson, quoted by Garnham (1949), consider that malaria at this age is an important cause of morbidity and mortality. Garnham, however, pointed out that he was working in an area in which malnutrition was uncommon and he suggested that the effects of malaria are conditioned by the state of nutrition. This suggestion may be the clue to many disputes concerning anaemia and infection in that if a patient is well nourished, a degree of infection can be withstood which could not be tolerated in a less well nourished person.

The malaria eradication scheme in Mauritius has provided evidence of the absence of any gross effect there of malaria upon the incidence of anaemia. Thus in 1948, 1,576 patients with malaria and 1,392 with anaemia were treated in hospitals in Mauritius, but in 1953, when malaria had been eradicated from the island and no cases had been encountered, 2,934 patients with anaemia were treated in these hospitals. Causes other than malaria are clearly here responsible for the common form of anaemia encountered and from these figures it may be doubted whether malaria plays an important part in the causation of anaemia among adults. From experiments carried out by McGregor *et al.* (1956) it appears clear that the effect of malaria upon the blood is most marked in the earliest years of life. These workers concluded, from a study of groups of children protected and unprotected against malaria, that in a hyperendemic malarial region by the end of the first two years of life unprotected children have acquired sufficient immunity to malaria to enable them to make good any ground previously lost in so far as weight, development and haematological values are concerned. These findings are consistent with the results of the observations made in Mauritius, which led to doubt as to whether malaria commonly plays an important part in the causation of anaemia in adults. Immunity and the state of the patients nutrition are without doubt factors of great importance in deciding whether anaemia is, or is not, caused, for as is well known, transient anaemia is produced by an attack of malaria even in well nourished persons if they have not any premunity to the infection.

BACTERIAL AND OTHER NON-HELMINTHIC INFECTIONS AS CAUSES OF ANAEMIA.

Bacterial and other infections are commonly overlooked as important causes of anaemia in the tropics. Indeed, some authors have considered them to be among the most common causes of anaemia generally (Batty, 1947; Diamond, 1950; Stergess, 1944). The anaemia produced by these infections is usually normocytic and normochromic, but it may become microcytic and hypochromic (Cartwright and Wintrobe, 1955). In patients with this type of anaemia there is a decreased rate of iron utilisation for haemoglobin synthesis, persistently low levels of iron in the plasma, diminished concentration of iron transporting protein in the plasma, a minimal rise in plasma iron following oral administration of iron and rapid removal of injected iron from the blood. From this it seems that the numerous common infections in the tropics must also be common causes of the common anaemias found there and more attention will have to be given to them in the future.

MEGALOBLASTIC ANAEMIAS.

Probably more work has been done on megaloblastic anaemia than on any other variety of anaemia in the tropics and this is understandable in view of the impetus to research which has been given by the successive discoveries of the action of haemopoietic factors in liver, of folic acid and vitamin B_{12}. The fascination of the aetiological and therapeutic problems which they present and the consequent interest which they evoke may have been responsible for the development of an exaggerated impression of their prevalence. At the present time it is common practice almost throughout the tropics for severe anaemias and for many mild anaemias to be treated with liver extract, vitamin B_{12} or folic acid without any attempt being made to examine the marrow and, in some hospitals, without any investigation being carried out other than a haemoglobin estimation. Not uncommonly no effort is made to determine the type of anaemia before treatment is commenced, but it may well be that the money spent on wasted medicaments would be sufficient to provide the additional staff needed for accurate diagnosis.

Once again accurate figures regarding the relative prevalence of different forms of anaemia in the tropics are almost completely lacking, but an impression of their frequency may be gained from the fact that, during a tour of India, Woodruff (1955*a*) personally examined and took notes on 186 patients with anaemia, in only 8 was there clear evidence of the condition being megaloblastic. Stott's work in Mauritius further indicates that megaloblastic anaemias are not there an important public health problem. It seems, therefore, that there is marked disparity between the great interest taken in them and the relatively small importance of the public health problem they present.

ANAEMIAS ASSOCIATED WITH PROTEIN MALNUTRITION.

There has been a tendency latterly to regard almost all anaemias in the tropics as being caused by deficiency of iron or by deficiency of, or non-availability of, specific haemopoietic factors, but to do so is surely to fall into the error of gross oversimplification. Much more than iron and specific haemopoietic factors are required for the production of red blood cells. They consist almost entirely of protein and protein deficiency is one of the commonest conditions encountered in the tropics. It would be very surprising indeed if protein deficiency did not play some part in the causation of the common anaemias in the tropics. Moreover, there is ample experimental basis for such an assumption and from the classical work of Whipple and his colleagues (Whipple and Madden, 1949) is derived the «concept of a large protein pool including plasma proteins and mobile cell proteins. The contributions to this pool derive largely from the liver and the withdrawal from the pool may concern any body cell needing protein or capable of storing some surplus protein. From this protein pool may be derived haemoglobin, plasma protein or cell protein. The circulating plasma is the medium of exchange and the body is solvent just so long as there are adequate protein supplies for any emergency».

A further link in the chain whereby protein malnutrition may lead to anaemia has been provided by Nizet and Robschiet-Robbins (1950), who in experimental work with dogs have shown the rate at which reticulocytes ripen to depend on the constituents of the plasma. They have concluded that the red blood cell at the reticulocyte stage is able to incorporate plasma protein into itself or to synthesise plasma proteins from amino acids. Thus if the plasma proteins are abnormal or deficient, some disturbance may take place in the ripening of reticulocytes and therefore in the formation of the mature red blood cells. Woodruff (1955c) showed that in children suffering from kwashiorkor the ripening of their reticulocytes was delayed.

Further experimental evidence of the parts that protein deficiency and liver damage may play in the production of anaemia is obtained from recent work carried out by Professor B. S. Platt (personal communication), who has shown that pigs kept on a low protein diet have considerably less haemoglobin than control animals and that those on a low protein diet with extra carbohydrates and in whom therefore there is a greater degree of liver injury have still lower haemoglobin values.

Whipple and Madden (1949) have stated that contributions to the plasma protein derive largely from the liver. In view of this, and of the part that these plasma proteins may play in haemoglobin synthesis, the widespread high incidence of liver damage among tropical peoples almost certainly plays a very considerable part in the production of the common anaemias there. It has to be borne in mind that albumin values in the serum of patients suffering from protein malnutrition may be quite fantastically low; thus of five children suffering from kwashiorkor and described by Woodruff (1955*c*) serum albumin values of less than 1 g % were encountered in three and the lowest value was 0.2 g.

It is, of course, difficult to recognise anaemia caused by protein malnutrition because very seldom does it present in pure form in man. When it does so, however, in experimental animals it is of a simple normochromic and normocytic type. In man concomitant iron deficiency often imposes its own characteristics upon it and leads to the anaemia present being considered to be the result of iron deficiency only. If, however, liver damage is associated with the anaemia of protein malnutrition, then target cells may be found in the peripheral blood and the red cells generally will be broader and thinner than normal. Cells of this type were described by Larsen (1948) and all physicians and pathologists who have dealt with patients with cirrhosis of the liver are familiar with them. Himsworth (1950) has reported similar broad and thin cells in rats suffering from methionine deficiency and liver damage. Cells of this kind have been found by Woodruff (1955*c*) to occur in the blood of children suffering from anaemia and kwashiorkor, in pregnant women suffering from anaemia and protein malnutrition and also in some adults similarly affected. These cells may give the blood the appearance of macrocytosis when a blood film only is examined, and they have certainly been the cause of confusion between anaemia associated with liver damage and that due to deficiency of specific haemopoietic factors.

Some doubt of the part played by protein malnutrition in the production of anaemia has been caused by the poor haematological response which commonly follows the administration of protein to such patients. Such doubts, however, are illogical for the anaemia here is unlike that of pernicious anaemia in which micro-quantities of haemopoietic substance only are deficient the other requirements of haemoglobin production being available. In anaemia caused by protein malnutrition the basic materials from which haemoglobin is made are defective or deficient and this abnormality can be corrected only slowly. There are probably two important causes for this slowness of response of anaemia in malnourished patients. Firstly the administration of protein to patients with liver damage may help to repair that damage, but may do so slowly and only when the liver's capacity to synthesise protein adequately has been restored can any improvement in the ability to synthesise haemoglobin be expected. Secondly, Elman *et al.* (1942) have shown that a deficiency of 1 g of plasma protein is equivalent to a deficiency of 30 g of body protein and that only after the total body protein deficit has been replaced can permanent elevation of the plasma protein levels be expected. Thus in a person of 70 kg in weight a deficiency of 1 g % of plasma albumen would be equivalent to an overall body deficiency of protein of approximately 1 kg. Bearing in mind the very low protein values encountered in some tropical peoples suffering from malnutrition, the overall bodily deficit in such patients can readily be understood to be enormous and must be replaced before permanent elevation of the plasma protein values can be expected. To such patients, a diet has to be given which provides basic requirements for protein and also makes a significant contribution to the deficit, and for this reason alone a high protein diet may be required for a very considerable period before any sign of improvement occurs.

There is a final speculative aside to which attention might be directed. If protein deficiency sufficient to impair haemoglobin synthesis occurred in the presence of an adequate iron intake — and the diets of most tropical peoples are adequate in this respect — then iron would be expected to accumulate in the liver and spleen and to produce haemosiderosis. Such a set of circumstances could explain the high incidence of haemosiderosis which is well known to occur among tropical peoples. With the object of testing this hypothesis I have recently carried out an experiment in which to one group of rats purified rations containing 30 % protein were administered and to the second group similar rations but containing only 6 % protein were given. To both groups the iron requirements were administered parenterally in the form of Imferon and after 10 weeks liver sections taken from control and experimental animals and stained with the Prussian blue technique have suggested that more iron is deposited in the liver of the protein deficient animals than in the controls. Woodruff (1955*c*) has emphasised the possible importance of protein malnutrition as a link between many aspects of pathology and altered physiology in the tropics and it is possible that this is yet another instance of such a link.

CONCLUSION

In conclusion therefore, the importance of anaemias in the tropics should be emphasised, for it appears that they may be responsible for very considerable morbidity, and especially among pregnant women, mortality. From the public health point of view, the problem of greatest importance is presented by the iron deficiency anaemias, aggravated in most cases by helminthic infection, but, although more difficult to delineate and rather more disappointing to treat, anaemia resulting from conditions caused by protein malnutrition probably presents a problem almost as great.

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RESULTADOS DO TRATAMENTO DA ANEMIA DA ANCILOSTOMÍASE

LUÍS TOMÁS DE ALMEIDA FRANCO

Desde que o carácter sideropénico da anemia da ancilostomíase ficou bem estabelecido pelos trabalhos de O. Cruz (1932, 1934), Rhoads e Castle (1932), Larizza e Ventura (1953) e Trincão e col. (1952, 1953) e que a simples desparasitação nem sempre consegue normalizar o quadro hemático, todos os esquemas terapêuticos utilizados têm como base a administração de ferro, a qual, por si só, sem qualquer procedimento anti-helmíntico, é geralmente suficiente para a completa restauração dos seus valores hemáticos.

Se bem que a terapêutica marcial seja habitualmente eficiente, alguns têm sido os casos em que foi verificada a sua falência, o que levou a pensar que esta seja devida à não desparasitação dos pacientes, apoiando-se a hipótese nos seguintes factos:

1.º A regeneração eritrocitária pode não compensar a espoliação do ferro que o organismo sofre desde que persistam as perdas de sangue ao nível da mucosa duodenojejunal, provocadas pela implantação dos vermes.

2.º A existência de alterações da absorção do ferro provocadas pelos seguintes mecanismos:

   a) Hipercinese gastroduodenal;
   b) Alterações da absorção do ferro que recentemente parecem ter sido comprovadas, com a utilização do ferro radioactivo, por Ventura e col. (1957).

3.º Aumento da deposição do ferro no sistema reticulendotelial, determinado por estimulação inespecífica, de maneira idêntica à verificada nas anemias por síndromas infecciosos.

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 518-534, 1959

Em continuação das investigações conduzidas sobre este assunto pela Secção de Hematologia do Instituto de Medicina Tropical, decidiu-se levar a efeito as seguintes observações:

A) Comparação da eficácia da terapêutica marcial em indivíduos anémicos portadores de *Ancylostomidae* nos quais se tinha prèviamente procedido à desparasitação com a obtida num outro grupo com graus semelhantes de anemia e carga parasitária, mas em que se mantiveram os parasitas, de modo a obter dados que nos indicassem qualquer acção dos vermes sobre a regeneração eritrocitária, e especialmente sobre a influência da manutenção das constantes perdas de sangue devidas às microlesões da mucosa intestinal.

B) Comparação dos resultados obtidos com a terapêutica marcial efectuada por via parentérica e por via oral, de maneira a evidenciar o papel das alterações da absorção do ferro, razão tantas vezes invocada para explicar quer a génese da anemia, quer ainda a falência da terapêutica.

Estas investigações foram levadas a cabo em indígenas da Guiné Portuguesa, com os quais constituímos os seguintes grupos:

1.º grupo: 12 indivíduos que foram prèviamente desparasitados pela tetracloroetilena, segundo o método preconizado por Carr e col. (1954), usando como critério de cura a negatividade de três exames de fezes consecutivos, pelo método de Willis, e cuja repetição foi realizada regularmente durante todo o período em que se efectuaram as nossas observações.

A este grupo de doentes foi instituída a terapêutica por administração intramuscular de um complexo Fe-Dextran (Imferon).

2.º grupo: 12 pacientes aos quais se fez o mesmo esquema terapêutico, mas sem prévia desparasitação.

3.º grupo: 11 pacientes nos quais a terapêutica foi conduzida por via oral, com sulfato ferroso na dose de 1,0 g diário (2 × 0,50 g), mantendo os parasitas e, consequentemente, as condições por eles originadas.

Em todos os doentes incluídos nos diferentes grupos foram efectuadas as seguintes determinações antes do início da terapêutica, cuja repetição foi feita a intervalos regulares durante todo o tempo em que os pacientes estiveram sob observação:

1 — Contagem de eritrócitos.
2 — Doseamento da hemoglobina: método fotocolorimétrico.
3 — Determinação do valor hematócrito: método de Wintrobe.
4 — Contagem de reticulócitos.
5 — Determinação dos seguintes índices: valor globular, hemoglobina média do eritrócito, concentração hemoglobínica do eritrócito, volume médio do eritrócito.
6 — Determinação da sideremia: método de Schales.
7 — Punção esternal: mielograma, ferro medular.
8 — Doseamento da capacidade fixadora do ferro pelo soro: método de Ventura.
9 — Doseamento da protoporfirina eritrocitária livre: técnica de Schwartz e Wikoff.
10 — Doseamento da coproporfirina eritrocitária: técnica de Watson.
11 — Doseamento das proteínas totais: método do biureto.
12 — Proteinograma: electroforese em papel.
13 — Determinação da velocidade de sedimentação: método de Westergren.

Pela observação dos resultados obtidos podemos concluir que:

1.° A normalização do quadro hemático se fez mais ràpidamente naqueles doentes que foram tratados pelo ferro administrado parentèricamente e com prévia desparasitação, o que parece apoiar o facto de que a espoliação de sangue provocada pelos vermes poderá constituir um obstáculo a uma mais rápida recuperação dos doentes. Em reforço desta opinião, existem os trabalhos de Brumpt e Song (1953), que pretendem demonstrar que o tratamento pelo ferro estimulará a actividade dos vermes, em consequência da qual aumentarão as perdas de ferro do organismo, pelo que poderemos pensar que a desparasitação abreviará possìvelmente a cura da anemia com esta patogenia.

QUADRO 1

		1.º grupo	2.º grupo	3.º grupo
Dias de tratamento		54 dias	31 dias	58 dias
Agente terapêutico		Imferon	Imferon	Sulfato ferroso
Dose total de ferro		2,830 g	2,310 g	12,2 g
Eritrócitos	Valor inicial	2 698 000	2 640 000	2 654 000
	Valor final	4 665 000	3 959 000	3 812 000
Hemoglobina	Valor inicial	4,98	4,45	4,93
	Valor final	10,95	8,99	10,27
V. globular	Valor inicial	0,65	0,6	0,6
	Valor final	0,8	0,8	0,9
Hgb. méd. erit.	Valor inicial	18,9	17,0	16,0
	Valor final	23,8	22,0	27
Hematócrito	Valor inicial	21	19,7	20
	Valor final	37,5	35	37,5
C. hgb. méd. erit.	Valor inicial	23	22	19
	Valor final	27	26	27
Vol. méd. erit.	Valor inicial	81	76	76
	Valor final	88	87	99
Sideremia	Valor inicial	45,7	26,8	23,3
	Valor final	60,3	93	43
Protoporfirina	Valor inicial	241,4	279,5	245,4
	Valor final	112,6	100,5	96
Transferrina	Valor inicial	500,0	399	328
	Valor final	61		
Ferro medular	Valor inicial	0	0	0
	Valor final	++	++	+−
Proteínas totais	Valor inicial	6,3	6,9	7,2
	Valor final	7,4	7,4	
Vel. sediment.	Valor inicial	54,8	41,9	53,7
	Valor final	38,6	49,7	35

2.º Quanto às alterações da absorção do ferro ao nível da mucosa intestinal, que Ventura e col. (1957) invocam para explicar uma das facetas da etiopatogenia do processo anémico, possìvelmente não são de fundamental importância quando se institui a terapêutica marcial por via oral e em que a quantidade de ferro ingerida é extremamente elevada. A apoiar esta hipótese existem os resultados obtidos por todos aqueles que têm experiência da administração do ferro *per os* no tratamento deste tipo de anemia, com os quais os nossos resultados obtidos no 3.º grupo são concordantes.

3.º Como facto de salientar, verificou-se que, apesar da intensa terapêutica, não se observou qualquer subida substancial dos níveis da sideremia. É do conhecimento geral que nas anemias sideropénicas, uma vez iniciada a terapêutica pelo ferro, se verifica numa primeira fase um aproveitamento muito maior deste metal para a síntese de novas moléculas de hemoglobina e que só mais tardiamente, quando normalizado o quadro hemático, se começam então a refazer os depósitos fisiológicos do ferro, e, consequentemente, a restauração dos valores da sideremia deve ser o resultado mais tardio desta terapêutica.

Levando este facto em linha de conta, foi nosso desejo assegurar aos pacientes uma maior dotação de ferro para que, ao regressarem às suas condições de vida habituais, os mesmos mecanismos que tinham ditado o aparecimento do quadro anémico pudessem ser enfrentados com maiores possibilidades de defesa, pelo que se decidiu fazer a administração de uma dose suplementar deste metal.

Deste modo, continuámos a terapêutica destes grupos, agora com o sulfato ferroso, *per os*, dada a impossibilidade de momento de usarmos um produto de administração parentérica.

Os resultados obtidos com esta 2.ª fase terapêutica constam do quadro 2.

Pelos resultados obtidos verifica-se:

1.º Os valores hemáticos não foram influenciados por esta terapêutica.

2.º No que respeita à sideremia, também não sofreu qualquer alteração, apesar das quantidades apreciáveis de ferro administrado.

QUADRO 2

		1.º grupo	2.º grupo	3.º grupo
Dias de tratamento		44	32	43
Agente terapêutico		Sulfato ferroso	Sulfato ferroso	Sulfato ferroso
Dose total de ferro		27 g : 5,67	31 g : 6,9	43 g : 9,6
Eritrócitos	Valor inicial	4 665 000	3 959 000	3 812 000
	Valor final	4 395 000	3 960 000	4 168 000
Hemoglobina	Valor inicial	10,95	8,99	10,27
	Valor final	11,48	10,1	10,30
V. globular	Valor inicial	0,8	0,8	0,9
	Valor final	0,9	0,9	0,9
Hgb. méd. erit.	Valor inicial	23,8	22,0	27,0
	Valor final	26,0	25,5	25,0
Hematócrito	Valor inicial	41,0	35	37,5
	Valor final	41,8	35,8	39,0
C. hgb. méd. erit.	Valor inicial	27	26	27
	Valor final	27	28,5	26,5
Vol. méd. erit.	Valor inicial	88	87	99
	Valor final	95	90	94,5
Sideremia	Valor inicial	60,3	93	43
	Valor final	57,6		51,5
Protoporfirina	Valor inicial	112,6	100,5	96
	Valor final	107		63
Transferrina	Valor inicial	61		
	Valor final	224		
Ferro medular	Valor inicial	++	++	+
	Valor final	+++	+++	++
Proteínas totais	Valor inicial	7,4	7,4	
	Valor final	6,8	6,7	
Vel. sediment.	Valor inicial	38,6	49,7	35
	Valor final	41,3	43	34

Dado que os resultados encontrados eram comuns aos três grupos, quer tivessem sido ou não prèviamente desparasitados, não se podia alegar que a manutenção das baixas da sideremia fossem provocadas pela existência de vermes no intestino e que, portanto, tivesse havido o aparecimento de uma situação de equilíbrio entre ferro ingerido/ferro perdido. Pensou-se então que esse facto fosse devido à não absorção do metal ao nível da mucosa intestinal, pois tem sido apontado por vários autores um sistema de bloqueio exercido pela mucosa intestinal reguladora da absorção, quando das menores necessidades do organismo, único meio de impedir uma hiperacumulação de ferro.

Para averiguar este facto fizemos uma prova de sobrecarga, administrando, por via oral, 1 g de sulfato ferroso, dose diária por nós habitualmente utilizada para o tratamento dos doentes, e observando seguidamente, às 2, 4 e 6 horas, as variações da sideremia (fig. 1).

Esta prova demonstrou-nos de modo cabal que se continua a absorver ferro, se bem que a sua absorção se faça em menor percentagem do que a que normalmente se encontra antes do início do tratamento. A observação da curva mostra-nos também que continua a existir uma marcada siderofilia dos tecidos, que é traduzida pela descida brusca verificada na 6.ª hora, se bem que, pela apreciação das quantidades de ferro administradas até essa data, elas fossem teòricamente mais do que suficientes para refazer por completo os depósitos normais de ferro.

Levando em conta que alguns autores afirmam ser necessária uma longa administração de ferro nas anemias em que se supõe existir um esgotamento dos depósitos, bem como a falta de dados concretos sobre a subida da sideremia após a normalização do quadro hemático, decidimos continuar a terapêutica e verificar o que se passava.

Esta nova fase da terapêutica foi iniciada com a administração de cinco injecções de 100 mg de ferro (Imferon), dadas em dias alternados, após as quais se efectuaram novas determinações da sideremia, a qual se verificou imediatamente ser, em todos os casos, mais baixa do que seria de prever pelos resultados referidos por Baird e Podmore (1954) (fig. 2), Cappel e col. (1954), Scott e col. (1954) e Cope e col. (1956). Seguidamente instituiu-se a administração de ferro por via oral, sob a forma de uma associação de ferro reduzido, carbonato ferroso e vitamina C, ao fim da qual se fizeram novas observações, cujos resultados constam no quadro 3.

Verificamos assim que os valores de ferro no soro não sofrem qualquer alteração depois de tão larga terapêutica marcial (fig. 3).

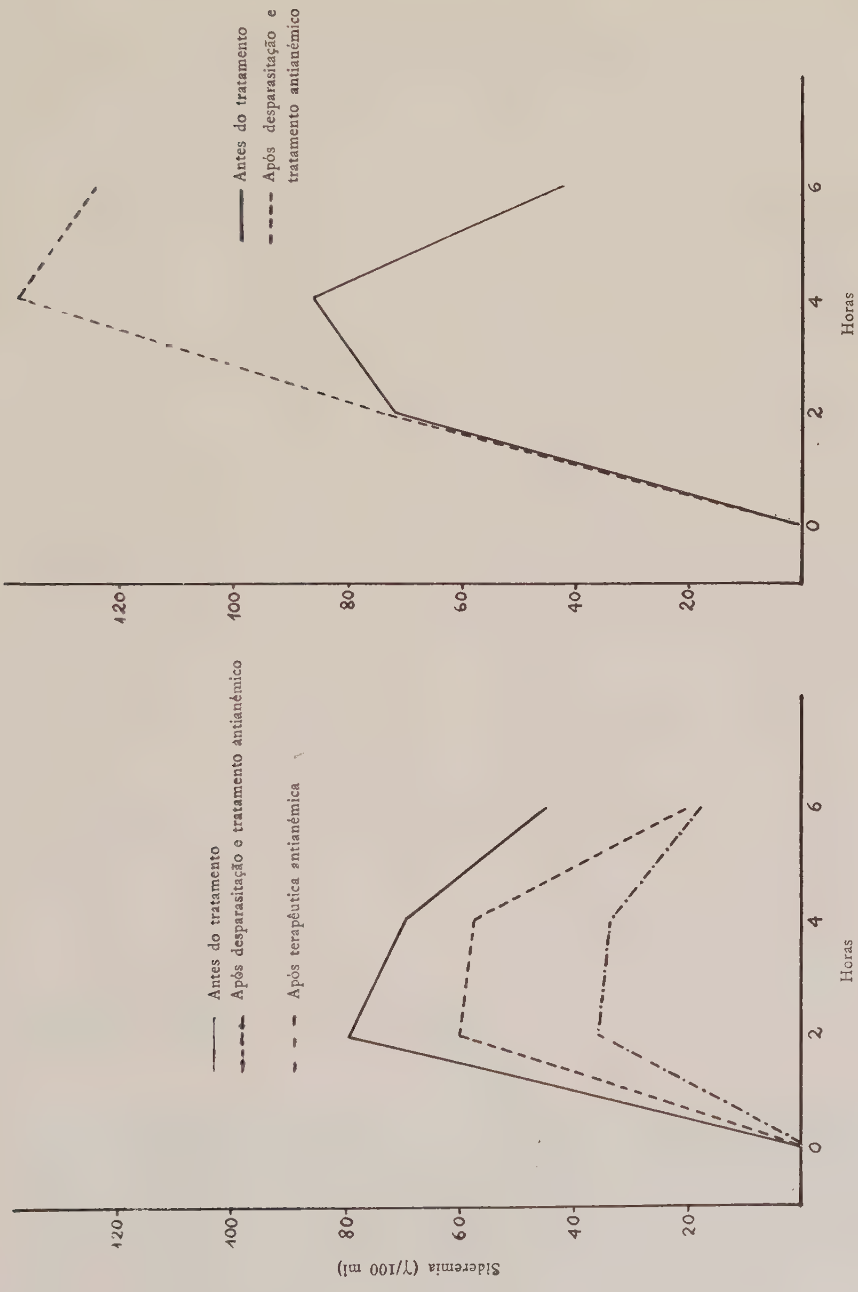

Fig. 1 — A, provas de sobrecarga realizadas nos doentes dos vários grupos; B, provas de sobrecarga em ancilostomados segundo Larizza e Ventura (1950)

QUADRO 3

		1.º grupo	2.º grupo
Dias de tratamento		41	88
Agente terapêutico		Fer-Cê	Fer-Cê
Dose total de ferro		0,50 I. M. 33,90 oral	0,50 I. M. 52,20 oral
Eritrócitos	Valor inicial	4 395 000	3 960 000
	Valor final	4 670 000	4 310 000
Hemoglobina	Valor inicial	11,50	10,1
	Valor final	11,40	10,4
V. globular	Valor inicial	0,9	0,9
	Valor final	0,8	0,8
Hgb. méd. erit.	Valor inicial	26,0	25,5
	Valor final	25	24
Hematócrito	Valor inicial	41,8	38,8
	Valor final	41,0	43,3
C. hgb. méd.	Valor inicial	27	28,5
	Valor final	28	27
Vol. méd. erit.	Valor inicial	95	90
	Valor final	88	91
Sideremia	Valor inicial	57,6	
	Valor final	58,4	43,3
Protoporfirina	Valor inicial	67,6	
	Valor final	109,8	81,0
Ferro medular	Valor inicial	+++	+++
	Valor final	++++	++++
Proteínas totais	Valor inicial	6,8	6,7
	Valor final	7,1	7,1
Vel. sediment.	Valor inicial	41,3	44,0
	Valor final	49,3	32,7

DISCUSSÃO

A anemia encontrada em 35 pacientes que albergavam parasitas da família *Ancylostomidae* era essencialmente sideropénica e a sua natureza encontra-se definida quer pelas características encontradas antes de iniciar a terapêutica, quer pelos resultados obtidos pela administração exclusiva de ferro como agente terapêutico.

Como facto principal a referir em relação às nossas observações, deve-se destacar que o ferro sérico dos pacientes apresentou uma fixidez, em valores anormalmente baixos, apesar de se ter feito uma prolongada e maciça administração desse metal. Quais as razões que podemos invocar para explicar tal comportamento?

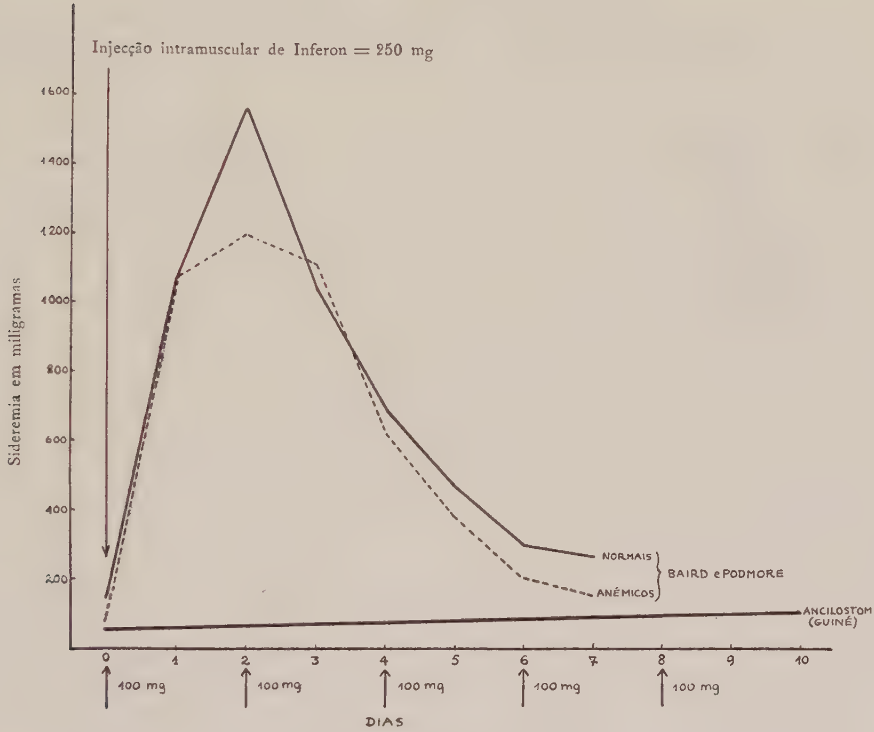

Fig. 2

1.ª A baixa sideremia ser de origem étnica: esta hipótese não pode ter qualquer validade, porquanto não só não seria natural tal facto, pois ele nunca foi verificado em África nem em qualquer outro ponto do globo, como também em observações anteriormente efectuadas em indivíduos dessa mesma região, por Trincão e col. (1950, 1952) e Janz e col.

(1957), se verificaram valores iguais ou mesmo superiores aos que se registaram nos Europeus.

2.ª Ser causada por uma falta de absorção do ferro: também esta razão não pode ser invocada por vários motivos:

a) No caso da administração parentérica do ferro ele é imediatamente colocado à disposição do organismo e, como

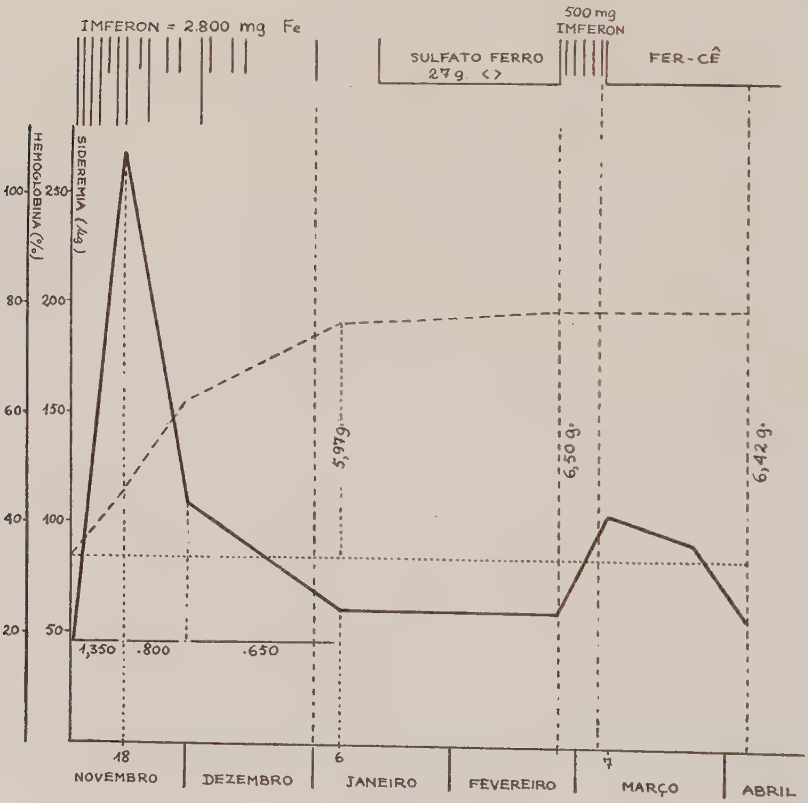

Fig. 3

não existe qualquer excreção apreciável, como Baird e Podmore (1954) verificaram, a sua utilização faz-se de maneira muito rápida, indo todo o metal introduzido por esta via armazenar-se nos depósitos fisiológicos, excluindo a quantidade despendida na hemoglobinogénese;

b) No caso da administração por via oral a sua absorção ao nível da mucosa intestinal foi demonstrada objectivamente pelas provas de sobrecarga;

c) Em todos os grupos se verificou uma regeneração eritrocitária intensa, que se seguiu à administração do ferro.

3.ª Resta-nos portanto a hipótese de que nestes casos tenha havido uma anormal deposição de ferro nos tecidos e que, por esse motivo, as doses de ferro administradas fossem muito maiores do que as teòricamente necessárias para o restabelecimento dos valores normais. Esta maneira de pensar tem a baseá-la o facto de que a pesquisa de ferro na medula óssea destes pacientes sofreu um aumento muito considerável, especialmente nas últimas fases das observações, sendo de prever um igual comportamento dos demais órgãos de depósito, visto que se tem verificado existir correlação entre o ferro medular e os restantes depósitos do organismo.

Temos ainda a apoiar esta nossa hipótese os seguintes factos:

a) A tendência verificada por diversos autores (Strachan, 1929; Gillman e col., 1945; Higginson e col., 1953; Walker e col., 1953; Edington e col., 1957; etc.) para a anormal deposição de ferro nos tecidos, muito especialmente localizada no fígado, em indígenas de diversas regiões de África (Bechuanalândia, Swazilândia, Zululândia, Basutolândia, Niassalândia, Tanganhica, Rodésias, União Sul-Africana, Angola, Moçambique e Costa do Ouro).

b) Investigações levadas a efeito por Pinniger e Hutt (1956), em coelhos, demonstraram uma marcada tendência para a acumulação do ferro, administrado sob a forma do complexo Fe-Dextran, nas células hepáticas, assemelhando-se bastante o aspecto histológico ao encontrado por Gillman e Gillman (1951) nas primeiras fases da siderose nos negros bantos da África do Sul. A confirmação de que este produto é ràpidamente armazenado no fígado foi levada a efeito por Grimes e Hutt (1957) utilizando o ferro radioactivo.

c) A verificação feita por diversos autores (Brock e Hunter, 1937; Widowson e McCance, 1937; Fowler, Barer e Spielhagen, 1937; Fowler e Barer, 1937; Gram e Leverton, 1951), citados por Walker e Arvidson (1953), de casos em que foi necessária a administração de doses extremamente elevadas de ferro para a normalização dos quadros hemáticos, o que traduzia uma fixação do metal muito além das suas necessidades.

d) O facto, por nós verificado, da diferença das variações da sideremia obtidas após a administração do Imferon em comparação com os valores obtidos por outros autores (fig. 4).

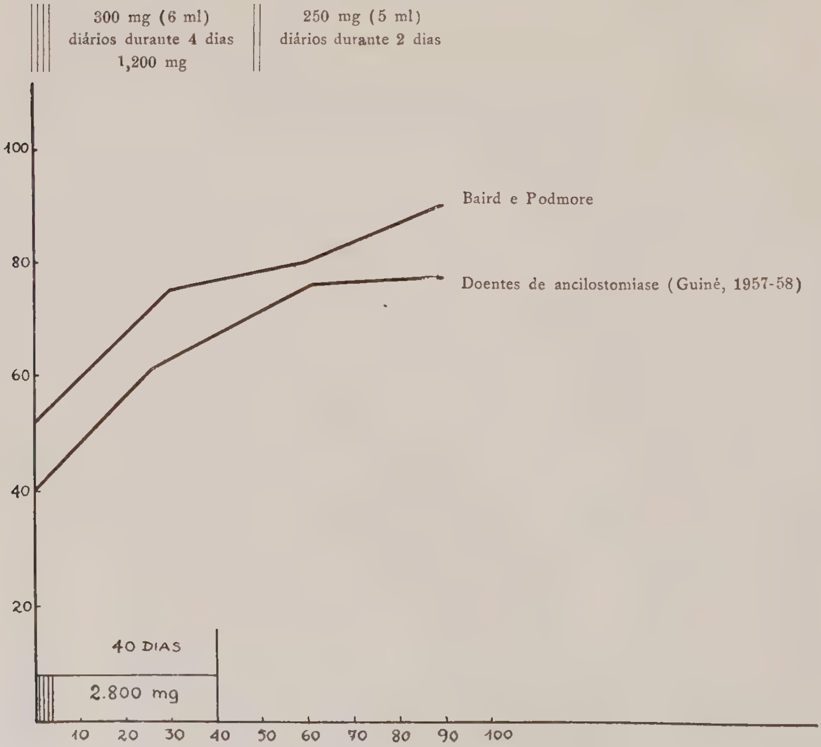

Fig. 4

e) Como corolário dos factos precedentemente apontados, e simultâneamente em seu apoio, devemos citar que em investigações anteriormente por nós levadas a efeito sobre a eficácia da terapêutica da anemia da ancilostomíase conduzida por via endovenosa, em paralelo com um outro grupo tratado por via oral, se obtiveram resultados que nos deixaram bastante perplexos e que passamos a apresentar:

QUADRO 4

	1.º grupo		2.º grupo	
Número de pacientes . . .	16		25	
Vias de administração . . .	Oral		Endovenosa	
Dias de observação	60–76 Média : 70		37–63 Média : 56	
Dose total de ferro	9,2–21,6 g 17,9 g		180–320 mg 297 mg	
	Antes	Depois	Antes	Depois
Eritrócitos	2 413 000	3 374 000	2 800 000	3 232 000
Hemoglobina.	7,0	9,2	7,7	9,0

Pelos resultados do quadro 4 somos forçados a admitir que os poucos benefícios sofridos por estes doentes se devem forçosamente à fixação do metal pelo sistema reticulendotelial, especialmente no caso da administração endovenosa (fig. 5).

Quanto às razões que poderão originar esta hiperavidez tecidular para o ferro, admitimos um provável hiperfuncionalismo dos órgãos do sistema R. E., hoje tão invocado nos indivíduos de raça negra e que se traduz pelo constante achado do aumento da velocidade de sedimentação e pelos quadros de disproteinemia, nos quais as hipergamaglobulinemias têm um lugar de relevo, como é do conhecimento geral.

Podemos pensar que as constantes agressões bacterianas e parasitárias que estes indivíduos sofrem provocariam uma excitabilidade anormal do S. R. E., como tendência do organismo para encontrar uma situação de equilíbrio entre o meio e as defesas orgânicas.

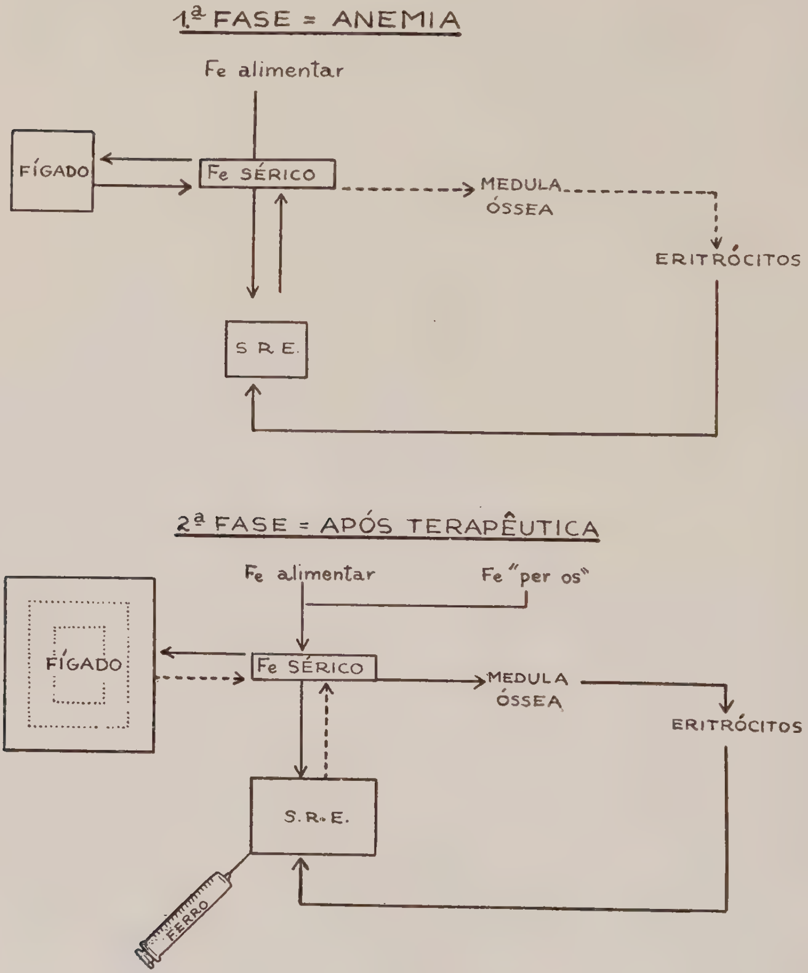
1ª FASE = ANEMIA
Fe alimentar
FÍGADO
Fe SÉRICO
MEDULA ÓSSEA
ERITRÓCITOS
S. R. E.
2ª FASE = APÓS TERAPÊUTICA
Fe alimentar
Fe "per os"
FÍGADO
Fe SÉRICO
MEDULA ÓSSEA
ERITRÓCITOS
S. R. E.
FERRO

Fig. 5

À nossa hipótese anterior poderemos acrescentar que a todos estes mecanismos talvez não sejam alheias algumas alterações metabólicas intracelulares em consequência de alterações crónicas da nutrição.

Como conclusão prática do que acabámos de expor, parece-nos poder assentar nos três factos seguintes:

1.º A conveniência da desparasitação prévia ou simultânea à terapêutica marcial.

2.º A conveniência em cessar a terapêutica marcial, quando de anemias sideropénicas em indivíduos de raça negra, logo que se normalize o quadro hemático, independentemente do nível a que se encontre a sideremia.

3.º A possibilidade da falência da administração endovenosa de ferro no tratamento deste tipo de anemia, em consequência da mais fácil fixação do metal quando administrado por esta via. Nos casos de maior urgência terapêutica as transfusões de sangue seguidas de administração de ferro por via intramuscular apresentam-se, portanto, como os métodos de eleição.

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THE ANAEMIAS OF AFRICA

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London, S.W.1

The chief anaemias to be considered are:

1. Secondary anaemias.
2. Iron deficiency anaemia.
3. Megaloblastic anaemias.

Abnormal haemoglobins causing anaemias:

S haemoglobin.
C haemoglobin.
Thalassaemia.

1. SECONDARY ANAEMIAS.

There has been little new in this field except that the influence of hook-worm disease and of malaria is now thought to be greatly exaggerated.

2. IRON DEFICIENCY ANAEMIA.

This is by far the most common type of anaemia in the low-lying, moist, tropical areas. 67 % of these patients are women. It has been suggested that the high incidence in these areas might be related to excess dermal loss of iron. The typical blood picture shows decreased size of the red cells and a low MCHC, whilst the marrow

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reveals normoblastic hyperplasia. Though iron greatly improves the condition, it has been found that in a proportion additional folic acid is required before the blood returns to full normality.

3. MEGALOBLASTIC ANAEMIAS.

Much of the recent work has been done on these anaemias and a better understanding has resulted from cobalt tagging of Vit. B_{12} with estimation of the content of stools, urine, serum and the liver. By estimation of the serum Vit. B_{12} and noting the response to therapeutic agents and the above it has been possible to differentiate the following three main groups:

a) Those responding to folic acid. These show normal serum Vit. B_{12} levels (300-1,300), normal absorption of cobalt tagged Vit. B_{12} from the intestine, normal gastric acidity, giant stab cells and megaloblasts in the marrow. They do not respond to Vit. B_{12} or to antibiotics, but respond to folic acid 5-25 mg for 5-16 days.

b) Antibiotic responding and oral Vit. B_{12} responding megaloblastic anaemias. These show low serum Vit. B_{12} (20-100), normal absorption of cobalt tagged Vit. B_{12}, normal gastric acidity and respond to oral Vit. B_{12} and to oral antibiotics (penicillin). It might be thought that these were true nutritional anaemias. Unfortunately, however, their incidence bears no correlation with diet. It has been noted also that this type of anaemia is uncommon with gross protein malnutrition such as kwashiorkor and neither does it bear any correlation with liver changes. It has been suggested that the high carbohydrate intake of these people may encourage the growth of certain intestinal bacteria which are either inimical to the synthesis of Vit. B_{12} or more probably compete with the body for it. Moreover when the intake of Vit. B_{12} is marginal, synthesis of Vit. B_{12} in the gut may be much more important than in better fed communities.

c) Megaloblastic anaemias responding only to parenteral Vit. B_{12} and like true pernicious anaemia. All these cases were in women and they showed low serum Vit. B_{12} levels, diminished absorption of cobalt tagged Vit. B_{12}, a histamine fast achlorhydra and they did not respond to oral Vit. B_{12} or to antibiotics. These megaloblastic anaemias were seen commonly in higher areas (3,000-6,000 ft.). Before frank megaloblastic changes had occured in the marrow there was macrocytosis in the peripheral blood. As an index of incidence in 57 consecutive cases 33 were found with megaloblastic marrows and 24 with normoblastic marrows. Again it is more common in women. This type of anaemia seems much less common in India.

THE ABNORMAL HAEMOGLOBINS.

Points of interest:

S and C haemoglobins are the common abnormalities in Africa. The home of the latter, which is much more localised, is the Northern Territories of the Gold Coast.

The chemical difference in these haemoglobins lies in the substitution of a valine radical for glumatic acid radical in S and a lysine for a glumatic acid radical in C.

The prevalence of S in spite of the lethal characteristics of the homogenous state seems to be due to the relative protection of infants carrying the trait (AS) from hyperinfection with falciparum malaria before acquired immunity develops. Cases of apparent sickle-cell anaemia (SS) may in fact be a combination of the trait with thalassaemia minor and the estimation of haemoglobin A2, which has the same electrophoretic mobility, as E is crucial in the diagnosis. An excess in foetal haemoglobin is common in true sickle-cell anaemia.

Cases of sickle-cell anaemia do occasionally survive to adult life when the anaemia does not seem to be severe and may be missed. Such cases are liable to sickle-cell crises after operation for say a salmonella osteomyelitis, to which they seem unduly susceptible.

About 15 % of our W. African troops carry the S trait (danger of high flights).

CC is uncommon and AC appears to be innocuous.

SC is quite common among the W. African troops and several are boarded out of the service every year because of this condition. Recurrent polyarthritis with recurrent episodes of haemolysis often following fever (malaria) or strenuous exercise is the usual clinical presentation. Haematuria in these men is not uncommon. Women with this condition are liable to sickle-cell crises and death during the last three months of pregnancy. These men should not fly above 4,000 ft. in unpressureised aircraft, as they are then liable to splenic infarction.

GENERAL DISCUSSION

Dr. L. G. Goodwin (U.K.) — The importance of hookworms on a contributory cause of anaemia is shown by a type of case well known to physicians in Ceylon. Dr. Standen and I have recently been to Ceylon and were impressed by the number of children from rural areas which had escaped mass-treatments with tetrachloroethylene, who were admitted to hospital in a critical condition. They had severe anaemia (Hb less than 39 %), gastroenteritis, and the stools contained large numbers of hookworm ova.

They were badly nourished; a child of 5 years of age often weighed only 16-20 lbs. Blood transfusions, iron by intramuscular injection and by mouth, intravenous fluids and antibiotics for intestinal infection usually brought them out of danger. The diarrhoea frequently persisted, and was only brought under control when the hookworm infection was eliminated.

It is not satisfactory to treat patients in such condition with large doses of tetrachloroethylene. We used a new drug called *Alcopar* (Bephenium hydroxynaphthoate), which has the advantages of being less toxic. It is not absorbed significantly, it can be given repeatedly and no preparation of the patient or purging is necessary. The drug also expels roundworms.

After treatment, the diarrhoea ceased and the patients began to benefit from a good diet and iron therapy. They recovered more rapidly than children treated with tetrachloroethylene.

In answer to a question by Prof. S. Adler, I told the meeting that the species of hookworm was *Necator americanus*.

Dr. H. Teixeira de Sousa (Portugal) — Inquéritos alimentares feitos em ilhas de Cabo Verde livres de ancilostomíase têm mostrado que há uma ingestão suficiente de ferro por parte das res-

pectivas populações, mau grado a existência de muitas anemias de carácter ferrigénico.

Os clínicos têm encontrado muita dificuldade em colher bons resultados com a terapêutica marcial, havendo portanto necessidade de estudar melhor o metabolismo do ferro nas mesmas populações.

Acrescento ainda que os referidos inquéritos alimentares mostraram haver uma ingestão muito pequena de Vitamina C e proteínas de origem animal.

ROUND TABLE ON ABNORMAL HAEMOGLOBINS

A round table discussion on abnormal haemoglobins was held on September 8th, presided over by Dr. H. Lehmann (U.K.), with Dr. R. R. Gouveia as Secretary. The following members of the 6th International Congresses on Tropical Medicine and Malaria were present:

R. J. R. Cabannes (France).
N. C. Ferreira (Portugal).
F. B. Livingstone (U.S.A.).
R. Modica (Libya).
A. P. Tompkins (Nigeria).
C. P. Trincão (Portugal).
J. C. White (U.K.).
A. W. Woodruff (U.K.).

The following resolution was passed unanimously:

It is recognised that the haemoglobin diseases present a major health problem in the countries where they are found — most of them tropical. Millions of people are affected in Europe, Asia, Africa, and the Americas.

With the expected decrease of infectious diseases, the importance of genetical disorders — amongst which the haemoglobinopathies are foremost — will become greater rather than lessen.

The encouragement is desirable of surveys of the distribution of thalassaemia and of the abnormal haemoglobins. Though much has been done, there are still wide gaps in our knowledge. Without such information, planning of public health measures will be handicapped.

With the rapid rate of discoveries of new haemoglobins and new disorders a real risk arises that the nomenclature of the haemoglobins will become confused. This gathering of workers interested in the haemoglobinopathies hopes that sooner rather than later steps will be taken to arrive at international agreement. As regards the terminology of clinical disorders cognizance is taken of the Recommendations for Terminology of the hereditary haemoglobinopathies involving haemoglobin variants prepared by the Colonial Medical Research Committee Working Party on sickle-cell trait and sickle-cell anaemia, London, and published on May 25th, 1957, by the *British Medical Journal* and *Lancet*.

THE DISTRIBUTION OF SOME RARE HEMOGLOBINOPATHIES IN LIBERIA

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During the past two years my associates and I have conducted a hemoglobin survey of the native tribes of Liberia. In this period paper electrophoresis has been done on some 2,000 bloods, fetal hemoglobin determinations on about 1,500, and moving boundary electrophoresis, solubility determinations, and other tests, on fewer numbers.

Hemoglobin S (sickle-cell hemoglobin) was found in approximately 7 % of all subjects, but the frequency varied considerably among the different tribes. In northwestern Liberia the tribes have 15-20 % hemoglobin S carriers; in central Liberia the frequency is about 12 %; and in southeastern Liberia it is less than 1 %. Solubility tests were done on most of the bloods which showed an AS pattern on paper electrophoresis and only a single one had the solubility of hemoglobin D. Thus, hemoglobin D seems to be somewhat rarer in Liberia than in the American Negro, among whom Chernoff found 4 hemoglobin D carriers out of 1,000. In contrast to other parts of West Africa hemoglobin C was also found to be quite rare in Liberia; only 8 Liberians with hemoglobin C being found in the

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entire survey. In addition to these widespread hemoglobins our survey encountered three rare hemoglobinopathies.

The first two of these are abnormal hemoglobins which migrate faster than normal hemoglobin in an alkaline buffer. When first discovered, they were called Liberian 1 and Liberian 2. Since then several attempts have been made to determine their relationship to other fast-moving hemoglobins which have already been assigned letters. Liberian 1 is similar to hemoglobin J, but at present does not appear to be identical to it, so that Agar and Lehmann have suggested assigning it the letter N. Liberian 2 now appears to be identical to hemoglobin K, which has been found by Cabannes in Algeria and by Lehmann and Edington in Ghana and elsewhere.

Hemoglobin N was found 8 times in the 2,000 samples for an overall frequency of 0.4 %. However, in the Mande tribes of the interior of central Liberia it had a frequency of about 2 %. A single other example was found in the Gola tribe for a frequency of 1 %. Since the Gola border on some of the Mande tribes which have higher frequencies, this finding could very easily be due to mixture. It appears that hemoglobin N is restricted to several tribes in central Liberia and is not found in the tribes in the north and south of the country. In addition Allison did not find it in Sierra Leone nor has our group found it in the Ivory Coast to the east of Liberia.

Hemoglobin K was only encountered twice in our survey and both these persons came from tribes in central Liberia.

Because of the rarity with which these abnormal hemoglobins are found throughout the world, it would appear that the mutation rates are very low. This is more evident when one considers that the heterozygote for either of these genes and the normal allele does not have any pronounced clinical symptoms, and thus appears to have normal fitness.

The distribution of hemoglobin N in several tribes at about the same frequency seems to indicate that this hemoglobin is an ancient inheritance of the peoples in this part of Liberia. This also seems to imply that the hemoglobin N gene does not have a very great selective advantage associated with it, since, if it did, one would expect it to have attained a higher frequency in this time. Thus, the value of about 2 % which is found among these diverse tribes would appear to be the highest the equilibrium frequency could be. In this way this gene, and most likely the gene for hemoglobin K, are not like the genes for hemoglobins S, C, E, and thalassemia which are found in rather high frequencies in some parts of the world.

The third hemoglobinopathy which our survey discovered seems at present to be different from the two previously discussed, since it seems to attain high frequencies. Among the Kru-speaking tribes of eastern Liberia, we have found several adults who have increased amounts of fetal hemoglobin which in some cases is as high as 45 %. Clinically these individuals do not seem to have symptoms associated with this condition, although, as is usual in Liberia, their hemoglobin values are below what is considered normal in Europe or the United States. In four tribes of eastern and central Liberia the frequency of adults having more than 10 % fetal hemoglobin was about 1 %. In the Webbo tribe the frequency was found to be 4 %. Also in the Grebo and Webbo tribes we found that 33 % of the individuals tested had greater than 2 % fetal hemoglobin, which is usually considered the upper normal limit in Europe or the United States. In the tribes of central Liberia 15 % of the individuals tested had greater than 2 % fetal hemoglobin, while in northern Liberia it was only 8 %. Other studies of increased amounts of fetal hemoglobin in adult Negroes, which have been done in Jamaica and East Africa, indicate that it is inherited as a dominant trait. However, we have now found evidence for thalassemia in Liberia and are not sure of the relationship of this high fetal trait to thalassemia. Further studies are now in progress, but it now appears that a thalassemia like gene is quite common in Liberia, particularly in thoses tribes with very low frequencies of the sickle-cell gene and high frequencies of this high fetal trait.

SECTION 6

TROPICAL HYGIENE & SANITATION

SUBSECTION 1

SANITARY EDUCATION

1ST SESSION: *Tuesday, September, 9th — 3:00 to 4:30 p.m. Room B*

The meeting was held under the chairmanship of Dr. E. H. Hinman and the Rapporteur was Dr. Hortênsia de Hollanda. The Secretary was Dr. Francisco Freire Júnior.

The following papers were presented and discussed.

A CONTRIBUIÇÃO DA EDUCAÇÃO SANITÁRIA NO *CONTRÔLE* DA ESQUISTOSSOMOSE E OUTRAS PARASITOSES E SEU PAPEL NO DESENVOLVIMENTO DAS COMUNIDADES

HORTÊNSIA DE HOLLANDA

Departamento Nacional de Endemias Rurais

RESUMO

O trabalho discute problemas e métodos de educação da população para o *contrôle* das verminoses, particularmente a esquistossomose, em áreas altamente endémicas.

Diante do custo elevado e do carácter temporário dos actuais métodos de combate aos moluscos vectores, bem como das limitações da quimioterapia para tratamento em massa, os programas de *contrôle* esperam da educação sanitária a mudança dos comportamentos que se relacionam com o contacto com focos e com o despejo dos dejectos humanos no solo e nos cursos de água, visando a eliminação ou redução das oportunidades de transmissão da parasitose.

A autora focaliza a ligação desses comportamentos, no Brasil, com: 1) os padrões de ajustamento ao meio ambiente; 2) a organização socieconómica e os modos predominantes de ganhar a vida: 3) as concepções da realidade e o

sistema de conhecimentos e ideias sobre a natureza; 4) as formas de relacionamento dos diferentes grupos sociais; 5) a mentalidade e a conduta peculiar das classes dominantes.

Os dados apresentados referem-se a comunidades onde a esquistossomose e outras verminoses figuram entre os mais graves problemas de saúde, com elevados índices de infecção humana, situadas em áreas insuficientemente desenvolvidas.

O trabalho realizado baseou-se nos princípios de educação sanitária da comunidade, em sua concepção de conjunto de processos destinados a ajudar as populações a definirem e a solucionarem seus problemas básicos de ajustamento ao meio ambiente.

A reestruturação dos comportamentos condicionantes da esquistossomose e verminoses processou-se, não apenas sob o impacto de novos conhecimentos e concepções da natureza, mas, essencialmente, motivada pela participação psicològicamente satisfatória em actividades socialmente prestigiadas, através das quais a população se desenvolveu e a comunidade se organizou para a solução de seus problemas básicos.

O relatório ilustra as possibilidades dos métodos empregados na redução dos índices de prevalência das parasitoses, apontando também as limitações impostas pelas circunstâncias económicas das áreas trabalhadas e pela estrutura, recursos e orientação dos serviços de assistência médica e educacional nessas áreas.

THE EXPERIENCE OF SANITARY EDUCATION IN THE FIGHT AGAINST DISEASES IN TROPICAL REGIONS

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In tropical regions one has to fight against diseases whose main origin is environmental and therefore preventable. In many cases knowledge of hygiene and the environmental conditions favourable to the spread of these infections is most useful and helpful in dealing with the problems of these diseases. Indeed once the various causes and favouring factors of disease are made known to the people and they are shown the value of hygiene, the need for control of environmental factors, and the necessity of prophylaxis, the battle against these diseases is half won. In one word, sanitary or health education is of considerable help in the fight against tropical diseases.

We are aware that in dealing with sanitary or health education it is necessary to know how it is to be propagated among the people whom it is desired to help. In this respect it is necessary to know the channel of approach or medium of getting in touch with the people so as to get a hearing. The co-operation of someone whom they respect or in whom they repose confidence is most essential if we are to achieve success. We must remember that some of the new teachings may clash with the customs and beliefs of the people and, unless someone close to them shares in the new outlook, it is difficult to effect changes, and for sanitary education to succeed.

Most tropical areas are rural, and in these rural areas there are usually local heads or chiefs whom the people regard as their natural leader and to whom they look for guidance. In addition there is usually a group of elders who help the local head or chief in admi-

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nistration and give advice when matters of importance are discussed. It is therefore of strategic importance that the first channel of approach should be through these chiefs and their local advisers. We have found this an important first step of approach in rural areas in Nigeria. When the support of the chief and local councillors is obtained, the ground is paved for further advance and the people are at least likely to listen to and ponder on what is taught. Once the co-operation of the people is gained and the barrier of antagonism broken down it becomes easy to advance in sanitary or health education.

Next in importance we find it useful to get the support and co-operation of the local religious leaders of all denominations, school masters, leaders of groups, indigenous organisations and societies, and youth organisations where these exist.

In a number of tropical countries such as in the Western and Eastern Region of Nigeria, and other parts of Africa, women have a say in the Government and they play important part in the life of community. In such countries sanitary education achieves success best when the interest of the women is roused to help the health programme.

The WHO Expert Committee on Health Education of the Public[1] in their first report said that education in any subject must focus on goals which seemed to the people being educated to be important and realizable within their capacities and resources. This point is aptly true of sanitary education. Once we choose a desired end which is important and realizable to the people, and we achieve success in carrying the people along with us in reaching our objectives, it becomes considerably easier to obtain ready support in other fields of sanitary endeavours.

In many parts of the tropical world procreation is regarded as one of the most important functions of life. The young mother treasures her child. To her motherhood is of great importance and significance. She can ensure the preservation of the life of her child, which is part of the goal of her ambition, by learning in the child welfare clinic how to care for the child, how to feed it properly, how to seek medical aid in sickness, how to observe the simple rules of hygiene, and how to prevent the child falling victim of malaria, that deadly enemy of young children. She can learn many other useful facts. The desire to preserve the life of her child spurs her on and the capacity for realising this end is there.

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[1] Expert Committee on Health Education of the Public. First Report WHO tech. Rep. Ser. 1954, 89.

She can get, through health education, knowledge about malaria and how to prevent it; and she can see that all the unnecessary foci of mosquito breeding in or round her home are destroyed.

It is necessary to remember that in dealing with sanitary or health education of the people in an area a thorough knowledge of the people is necessary. We must know their ways of life and customs, their beliefs and superstitions, their needs and resources, because it is only by working through knowledge of these facts that real success can be achieved. In relation to beliefs, for example in a community such as in Western Nigeria where it is already believed that flies have connection with transmission of Yaws, it is easy to graft on this basis the explanation of transmission of other infections by flies through contamination of food. Also in the same area where it is believed that sputum must be covered with sand or disposed of in the latrine to prevent its being used for malicious spread of tuberculosis, we have the basis for teaching hygienic habits which can be extended to include prevention of a large number of diseases common in tropical regions. Obviously if it is undesirable for sputum to be exposed, but better for it to be disposed of in the latrine, surely the next corollary is that the latrine itself must be protected and kept in good condition to prevent the spread of helminthic and other diseases. There must be numerous examples of similar beliefs in many parts of the world which can be used as basis for health education.

In their first report referred to above the WHO Expert Committee on Health Education of the Public defined the objectives of health education as follows:

> The aim of health education is to help people to achieve health by their own action and efforts. Health education begins therefore with the interest of people in improving their conditions of living, and aims at developing a sense of responsibility

> for their own betterment as individuals and as members of families communities or government.
>
> Health is but one of the elements in the general welfare of the people, and health education is only one of the factors in improving health and social conditions. It is, however, an indispensable factor and should therefore be integrated with other social economic, health and educational efforts.

The first part of this quotation shows the need for educating people to achieve health through their own actions and efforts. This is a very important point. The people must want, or be shown the need for, improvement of environmental health conditions and they must be educated in such a way that they desire to achieve this desired goal through their own effort. It is not enough to preach good sanitation, but we must so stimulate the people that they would not only be convinced but also desire to achieve good sanitary results by their own efforts. It is common human knowledge that what is achieved through one's personal efforts is much more appreciated, valued and lasting.

Therefore in sanitary education it is necessary to set goals which are important to the people and are capable of realization within their capabilities and resources, and moreover the people must be made sufficiently interested to wish to participate and achieve these objectives through self help.

As an illustration one may quote the case of the rural town of Ilora, some 141 miles north of Lagos, the capital of Nigeria, West Africa. In this rural town of 26,000 there were the usual high incidence of malaria and high parasitic infestations, malnutrition and high infant mortality. The infestation rate with *S. haematobium* was high. There was a swamp just to the north end of the town and it bred mosquitoes profusely and in addition there was a large population of the snail *Physopsis globosa,* the intermediate host of *S. haematobium*.

A survey of this rural town and others had been made previously. But the people in this town felt the need for a health centre so much that it became a goal which they believed is realizable through their own combined community effort. And by community effort they built themselves a health centre which is now staffed partly through their own locality authority and partly by the University College Ibadan, Department of Preventive and Social Medicine.

Through health education the people began to understand the relationship between malaria and the high infant mortality in their community; the relationship between malaria and debility and lack of energy; the relationship between haematuria and schistosoma infections and vague pains in the body. They learn the correlation between bad sanitation and worm infestation. And they also come to know that the swamp at the edge of their town was a menace to life because it bred mosquitoes and dangerous snails.

What must they do to save their babies' lives and to improve their own health? Is the goal attainable? Yes, it is attainable and within their capacities and resources to achieve, they were told.

Women listened to the Health Sister's advice about the feeding and care of their babies, about prophylaxis against malaria, about destroying all mosquito breeding sources and the need for supporting the sanitary officer in his efforts to improve hygiene and sanitation in their homes. They strongly urged their men folk to do something about the mosquito and physopsis ridden swamp and supported their efforts. And to-day that swamp no longer exists, it has been converted into a large fish pond about 3 acres. Mosquitoes no longer breed in it, the physopsis population has been reduced to negligible proportions, the fish in the pond provides protein to supplement the poor protein diet of the people, and the sales of the fish also formed new and additional source of revenue to the local authority.

The work of sanitary education continues in this area and more advance is being planned with the co-operation of the people and their local head Bale. And the women continue to take active part. The important role of women in health education and propaganda cannot be overstressed. They look after the home and its sanitation, they look after the children as well as the father, and they provide the driving force for those actions which they regard as necessary to be undertaken in the interest of the health of the family and of the community as a whole. The co-operation of women is essential to success in sanitary or health education particularly in those countries where they are vocal and progressive.

Health or sanitary education devolves largely on doctors, dentists, nurses, health visitors, district or community nurses, midwives, sanitary or health officers and auxilliary staff. These people must have sound knowledge of the people among whom they work. They must be patient and tactful so as to gain the confidence of the people, which is so essential to the success of any health education programme. A sound understanding of customs, beliefs and prejudices, and of the social, cultural and economic conditions of the people is most desirable if maximum benefit from health education is to be assured. Training in psychology and human relations is most useful and will help in enabling the sanitary educator to get closer to the people and their lives.

Sanitary or health education can be propagated through hospitals, dispensaries and health centres, and in all these places the doctors, nurses, health visitors, community nurses and midwives play important parts. Societies such as Mothers Union and youth organisation as well as schools and educational institutions form important centres for disseminating health education. Mass direction of attention of the public to sanitary education can be achieved during health weeks when a whole week is devoted to the preaching of health and display of matters related to the subject. In rural areas the association of health weeks with agricultural shows is usually successful. Farmers far and wide, who perhaps under ordinary circumstances might not come into town, have presented to them health education during the show of their products. In Nigeria such shows have proved most successful in advancement of sanitary education.

There is no doubt that sanitary education can help achieve much in the battle against disease in the tropics, where in many cases the economic condition is such that it is difficult to find the purse to meet all that is required to fight against these diseases. It has been stated above that many of these diseases are preventable, and if therefore with sanitary education we are able to reduce them, then the little sum available can be channelled to other health purposes. Sanitary or health education fulfils a most important roll in the fight against diseases in tropical regions.

PROBLEMS IN ORIENTATION AND TRAINING OF PUBLIC HEALTH PERSONNEL IN HEALTH EDUCATION WORK IN THE PHILIPPINES

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PHILOSOPHY AND OBJECTIVES OF TRAINING IN HEALTH EDUCATION

That health education is an integral part of all public health services and as such is an essential function of all health workers is a generally accepted fact. F. H. Sandford has stated the prophetic phrase:

> In the years ahead all the health and welfare professions will have to move along a road already being explored by public health. Down this road there will be *more education* and less priestly mandate, more advice and less control, more consultation and less prescription, more facts and fewer arcane pronouncements (7).

The Director General of the World Health Organization in 1951, in presenting his Programme and Budget Statement, enunciated:

> Experience has shown that when the main reliance is upon *legal* rather than *educational* measures, public response is often a mere passive compliance, or, at times, hostility. Thus health education of the public is not only a long-term objective in

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[1] The Author did not attend the meeting.

> itself but also an important complement to each of the health services in the general health programme, and the technique of health education is a part of every phase of public health administration (9).

Health education as referred to in these pronouncements is taken in its broader and modern sense. Quoting from The Expert Committee on Health Education of the Public of the WHO:

> The aim of health education is to help people to achieve health by their own action and efforts. Health education begins, therefore, with interests of people in improving their conditions of living and aims at developing a sense of responsibility for their own health betterment as individuals and as members of families, communities or governments (8).

This really means that when the educational approach is used in public health the goal is usually that of change — change of information, attitudes and behavior. Change of behavior takes place when an individual substitutes a scientifically more effective health practice for another one, or when an individual takes action which leads to more effective health practice (2).

This broader concept of health education is relatively new in the Philippines. True it is that public health literature may mention health education programmes and activities even in the more remote past (as far back as the late 1800's). However, these programmes and activities were mostly centered on the dissemination of health information through telling people what to do, through health campaigns and propaganda in the form of newspaper publicity, handouts, pamphlets, radio broadcasts, health contests, film showings, etc. By and large these were labeled as the whole of health education. Only within the more recent past have public health workers become increasingly conscious that the success of public health not only depends on an informed public but to a greater extent on how people change favorably their knowledge, attitudes, feelings and behavior.

Health education so conceived is a complex process:

> The public health workers desirous of helping people toward changed health behavior must have a thorough knowledge of the problems involved, that is, of the change required. He must have interest in and understanding of the people and the communities among which the change is needed, including their attitudes, beliefs, current practices and value systems. He must be aware of the possible effects of change on their life. He needs information on resources that can be used in bringing about the change and on ways of using these resources. He must understand the principles of health education and must have command of the methods appropriate to his field of work. He must know which methods are most likely to evoke desirable changes under different circumstances and a sensitivity as to when it is wise and right to use the methods (3).

It is obvious that in order to achieve the afore-mentioned goal it is essential to make provision in the training of all health workers for the study of and practical experience in health education which is in essence working with people. It is essential that all health workers be trained to make the best possible use of all health education opportunities and to possess both knowledge and skills in the use of educational methods, approaches, and media (8). This is particularly true for the Philippines, where, in the last few years, public health programs have been given a boost in the country's efforts to serve the rural areas. This has resulted in the unprecedented increase of public health workers and a growing consciousness for the need of additional skills to do more effective health education work.

While the need for public health training has been recognized in the past, this awareness became more pronounced with the rapid expansion of public health services. In recognition of these tremendous training needs, the Philippine Department of Health launched in 1953 an in-service training program on a wider scale in all phases of public health for all categories of public health personnel. These training programs are in the form either of graduate studies at the Institute of Hygiene, University of the Philippines, or of short-term training in regional training centers of the Department of Health or of continuing staff development programs on the job. This has been augmented by overseas study grants through the aid of international, bilateral and multilateral agencies. Health education is integrated into these different types of training programs of the Department of Health and is an elective course at the Institute of Hygiene. A more specialized kind of training is given for public health educators.

Briefly stated then, the objectives of this training in health education are: 1) to develop a deeper realization that health education is an integral part of public health services; 2) to create further awareness and develop an acceptance among public health workers of their health education opportunities and responsibilities; 3) to help public health workers do a better job of helping people towards desirable changed behavior through (*a*) a better understanding of themselves and people and (*b*) the development of more knowledge and skills in the use of educational methods, approaches and media, (*c*) the continuous evaluation of the educational aspects of health programs and (*d*) the ability to become more effective members of the public health team for effective health education.

TRAINING AND ORIENTATION DEFINED

At this point it may be necessary to dwell on the definition of the terms *orientation* and *training*. *Webster's New International Dictionary* defines *orientation* as «the act or process of orienting; adjustment to an ideal or a principle; determination or sense of one's position with relation to environment or to some particular person, thing, field of knowledge, etc.»; and *training* as «the act, process or method of one who trains; education, discipline, systematic drill [...] under supervision; state of being developed in strength or ability or proficiency». From the above definitions, it is easy to see the relationship between orientation and training; but difficult to delineate where one ends and where the other begins. While «orientation» may seem to be a necessary phase of training, «training», on the other hand, may be a necessary process to achieve a certain kind of orientation. For purposes of this paper, however, an attempt will be made to differentiate the two in terms of their general usage in the training programs heretofore mentioned.

Training as generally used may either be «academic» or «practical» or both. The one year academic training leading to a C.P.H. or M.P.H. degree is obtained at the Institute of Hygiene of the State University, where field training is usually integrated. The purely «practical field training» is provided for by the Division of Personnel Training and Health Education of the Department of Health and may last from 2 weeks to 8 weeks. The introductory phase of this «field training» program is usually referred to as the «orientation phase». This includes an over-view of the entire training content for the particular public health discipline and of the total public health program of the Department of Health and related official and non-official agencies including their organizational and administrative set-up. For the most part this is generally a one-way process where the «trainee» is on the «observing or listening end», but, to some extent, the trainees are involved in the planning of what is to follow. These are intended to establish a common frame of reference for all categories of trainees prior to the specialized phase of the training. «Training», on the other hand, includes a kind of specialization required for the particular discipline and generally consists of active participation in pertinent situations in the on-going public health services of the training centers based on the philosophy of «learning by doing».

PROBLEMS IN THE TRAINING AND ORIENTATION OF HEALTH WORKERS IN HEALTH WORK

1. BASIC PREPARATION OF HEALTH WORKERS WHO UNDERGO TRAINING.

Mountin has stated:

> Public health is an applied technology resting on the joint pillars of natural science and social science. For the past century, the natural science foundation has been magnificently strengthened — strengthened to the point where we now have the

> technical knowledge to eradicate or reduce greatly much of the misery to which man has been heir. Yet vast amounts of preventable or controllable disease and disability remain because the social science foundation is relatively weak. Until both the pillars of natural and social science are strong the arch of public health will not be firm (6).

This statement implies that much is yet to be desired in the basic preparation of public health workers in terms of social science requirements. Ideally, all health workers should have had a broad cultural background, with studies in the humanities, natural sciences and behavioral sciences (3).

While knowledge and understanding of the social sciences is an essential background for public health in general, it is especially significant for health education. If public health workers are to assume the responsibility of working with people towards desirable changed behavior, and if changing behavior is a complex process, necessarily public health workers must be equipped with some knowledge of psychology, education, anthropology, sociology, social psychology, etc., in addition to knowledge and understanding of public health courses and principles and methods in health education. In short, public health workers must draw heavily on the theory and research findings of the social sciences to do an effective health education job.

As is true in many countries of the world, a study of undergraduate curricula for physicians, nurses, public health engineers and public health dentists in the Philippines would show that in the past very little, if any, of the social sciences have been required. Many of today's trainees are drawn from the old graduates. They therefore come for postgraduate or practical field training with very meager, if any, background on the social sciences. Even if content materials may have been given, the manner in which they were taught may have contradicted the very philosophy of the social science courses. There may not have been any provision for cooperative planning and action, team work, interchange of ideas so that an acceptance, understanding and appreciation of people for what they are, as well as an understanding of the relationship of social factors on peoples' behavior may never have been experienced.

The problem therefore becomes one of integrating these basic ideas and principles in the training programs at a late stage — ideas and principles which should have been an essential part of their basic education.

This problem becomes more acute in the training of auxiliary health personnel like the midwives and sanitary inspectors, who have little, if any, professional training. Yet, these are the people who do most of health education work at the «grass roots» level.

2. A WIDE RANGE OF EXPECTATIONS OF HEALTH EDUCATION BY PEOPLE WHO UNDERGO TRAINING.

The expectations of people who come for training are as varied perhaps as their own professional goals, needs and interests; their own experiences and background; their own concepts of health education. These expectations generally range all the way from wanting to learn better ways and means of disseminating health information (one way process) to wanting to learn the one «best» method in health education with the hope that health education will serve as a panacea for all public health ills.

Based on the philosophy that as public health workers help people toward desirable changed behavior they should as «change agents» be willing to change themselves, the problem then is how to design a training program that would alter old perceptions and create new attitudes and insights. There is the added problem of trying to satisfy and, in other instances, help health personnel reexamine their expectations.

3. LIMITED AMOUNT OF TIME GIVEN OT HEALTH EDUCATION ESPECIALLY IN THE FIELD TRAINING PROGRAMS.

A study of the field training programs for the different categories of health personnel, except public health educators, will reveal that, by and large, health education sessions are scheduled from one-half to two-day sessions only. This is usually spent by having a balance of didactic and Socratic teaching. Through informal group discussions, buzz sessions, role-playing, report-back sessions, etc., an attempt is made to demonstrate some health education methods and techniques while putting across some basic concepts and principles in health education. This theoretical introduction is supplemented by observing health education activities in the field as they are integrated in the different public health services such as Maternal and Child Hygiene, Environmental Sanitation Control, Communicable Disease Control, etc., and as specialized activities such as community health organizations, study groups, health classes, etc.

In the academic training program at the Institute of Hygiene, health education courses are on the main electives. While health education principles and concepts are integrated in other courses, only those who elect the courses are introduced to health education formally. While it is felt that the inclusion of health education even as an elective course is already a big stride in public health, there is still room for improvement. The 24 hour elective course covering a school term (2-½ weeks) is rather short. Students in their evaluation of the courses have always recommended that more time be given to it.

On the basis of definitions given of *orientation* and *training* we can see that health personnel other than health educators actually only get an «orientation» in health education. The short time alloted for health education would limit the chance of changing or modifying long standing concepts and expectations. In some instances it may only tend to upset these established beliefs, which may be as correct to health workers as they are wrong to us; to confuse and at times to threaten them, which results only in frustration and hostility.

The problem then is: how can the curricula of the Department of Health Training Program and the Institute of Hygiene be so revised as to give more time for health education and make health education as one of the «core» subjects taking into consideration 1) the already heavy pressure of other subjects which seem equally important and 2) the limited amount of time allowed for the entire training program?

4. THE «TRAINER» IN HEALTH EDUCATION.

As in most countries, health education in the Philippines is still in its infancy. Only in 1950 was the first public health educator assigned in a health department set-up; only in 1952 was a six-year national programme in health education approved which entailed the employment of a handful of health educators at the health department; only in 1955 was a graduate course approved at the Institute of Hygiene; only in 1957 were the five regional training centers of the Department of Health manned with professionally trained health educators.

The task of orienting and training of health personnel in health education falls mostly on the health educators, while the rest of the training staff assume some degree of responsibility. Considering 1) the limited amount of experience of the health educators, they being relatively new in a young field, 2) the relative youth of the health educators compared to some health workers who have many years of experience behind them and 3) the academic qualifications of these new workers (Bachelor of Science in Education with a Certificate or Master of Public Health degree and without medical training), it can be understood why other public disciplines «raise their eyebrows» when these health educators assume the role of «trainers». Still, one graduate study of expectations which selected public health workers have of health educators revealed that they are expected to guide other public health workers in the selection of educational methods and approaches and to help improve their methods of teaching and dealing with people (1).

The problem then is: how can health educators best interpret their role and demonstrate their special understandings and skills so that they may be better accepted as specialists in their particular field and therefore more effective trainers?

5. THE ATTITUDE OF PUBLIC HEALTH WORKERS TOWARDS HEALTH EDUCATION AND HEALTH EDUCATORS.

In spite of the great care taken in the health education training programs to develop health education concepts, knowledge and skills as something distinctly separate from health educators, there seems to be a general tendency to think of one as inseparable from the other. It is always emphasized that there is a dearth of professionally trained health educators in the Philippines; that health educators cannot *supplant* but only help *strengthen, extend, supple-*

ment or *facilitate* health education activities of other health personnel. Nevertheless, there seems to be a general feeling that systematic and organized planning and implementation of health education programs *cannot* go on without a qualified professional health educator.

The problem then is: how can health personnel be trained in health education in order that they would accept health education as one of their basic functions and in order that they would assume their share of responsibility for leadership in the planning and implementation of health education programs with or without a health educator?

6. TRAINING IS FOCUSED ON THE LOWER RANKS OF HEALTH WORKERS.

Majority of health workers who undergo training are drawn from the municipalities and provinces, seldom from the upper ranks. There is of course some danger to such a practice. What may happen is that when the trainees return they do not see things eye to eye with their chiefs; they do not speak the same language. Whatever new ideas they would like to put into action, may not be understood or appreciated and may be blocked.

While this may appear mainly as an administrative problem, it has some implications for training. We believe that training is a continuous process and that the follow through is most important. If administrators block the introduction of new ideas, resulting frustrations would tend to dampen whatever interest trainees may have in improving existing services and in broadening their horizons.

The problem then is how to organize training programs so that they would become attractive to the upper echelons of public health workers, who in turn could lend the necessary leardership, guidance and support to those on the «firing line».

7. THE PROBLEM OF EDUCATIONAL ATTAINMENT, SOCIO-CULTURAL FACTORS AND COMMUNICATION IN THE TRAINING OF FOREIGN COUNTRY NATIONALS.

Public health knows no boundaries. The Philippines, like other countries, feel that it should extend its training facilities to those who want to avail of them. Within the last few years an increasing number of trainees have been sent for training from the different public health disciplines from the Southeast Asian and Western Pacific countries. The patterns of school systems are as varied as the countries from whence they come and present the difficulty of assessing their educational attainments. Then there is the difference in the socio-cultural background of the trainees and the problems of communications (many of the trainees can hardly speak English), which entail more attention and care in designing the training program so it could be tailored to fit the need of trainees in terms of their own educational background and «back-home» situations. It would seem desirable that the training officers need to be better acquainted with the conditions of the sending countries. In other words, there is need for training the «trainers».

The problem then is: how can we do a better job of extending our training facilities to foreign country nationals taking into consideration socio-cultural factors in their back-home situations, educational qualifications, communications problems, and the «trainers'» own limitations without sacrificing the quality of training for our own local personnel?

8. THE NEED FOR MORE RESEARCH ON THE APPLICATION OF KNOWN AND SOUND SOCIAL SCIENCE PRINCIPLES TO HEALTH EDUCATION AS WELL AS THE NEED FOR MORE TOOLS IN RESEARCH.

As has been previously stated, health education will have to lean heavily on the findings of the social sciences. Fortunately more behavioral scientists have started to work in the field of public health and we see an increasing number of studies of significance to health education. There are many studies focused on perception and how they affect human behavior; on socio-cultural factors as they affect health; on mass persuasion and communication; on group dynamics. While useful studies have been undertaken, there is still so much to be done, among which is research on the application of these findings to health education. Sandford said:

> I have a strong suspicion that there now lies on library shelves a good deal of material in psychology — and in the

> behavioral science — that could, if somehow made properly available, be of genuine usefulness to you. Somehow it just lies there, immobilized in generalization and hidden by a screen of technical language. This material needs to be put into such a form that you could tell whether or not it has relevance to you (7).

Or, to quote from Knutson on the same subject:

> ... we must find better ways of discovering how health and public health issues fit into the private worlds of people. For example, how do people perceive their chronic symptoms? What personnal and social cues lead them to act early so that preventive measures can be taken?
>
> There is a need also for further research to learn who the various groups in the community identify as their trusted communication «gatekeepers». What are the informal channels through which they obtain new ideas? Who do they see as the experts on problems relating to health? Where do these experts seek advice? (5).

And we may add: what make people carry their decisions into action?

Corollary to these we need more and better tools for research. Through such researches we can make more contributions to public health and make the content of training programs more scientific as well as realistic.

The problem then is: how can we stimulate more research in partially tilled or untilled fields in order to shed more light on many issues which would make training in health education more sound, scientific and useful in public health practice?

These then are the paramount problems in the training and orientation of health personnel — physicians, nurses, public health engineers, public health dentists, midwives, and sanitary inspectors — in health education. This paper does not include the problems in the training of health educators as a separate discipline even though some of the problems discussed do apply to their training. The particular problems of training health educators may merit a separate paper.

While the above problems may seem numerous and difficult, they certainly are not beyond solution. Positive steps have and are being taken to overcome these problems to ensure a brighter future for health education in the Philippines!

SUMMARY

The paper is presented in three parts. The first part discusses the philosophy and objectives of training in health education of health personnel in the Philippines. The second part attempts to differentiate between *orientation* and *training* as generally used in the training program. The third part presents and discusses the problems encountered in the Philippines. These problems may be summarized thus.

1) The basic preparation of health workers does not include enough social science courses as to enable them to understand and appreciate basic health education concepts at this late stages of their professional training.
2) The wide-range of expectations of health education by health workers are by and large not in consonance with basic health education principles and often serve as a block in imbibing these principles.
3) The limited time alloted for training in health education limits the chances of changing or modifying long standing concepts and expectations.
4) The health educators who assume the major bulk of the responsibility for training in health education do not have enough experience and the necessary status to be accepted as «trainers» by other public health disciplines.
5) Public health workers cannot seem to be able to dissociate health education from health educators. The latter are expected to relieve them of their health education responsibilities.
6) Training is focused on the rank and file of public health workers and health workers do not get the necessary guidance and support from their administrators in carrying out whatever new ideas they have acquired.
7) The educational attainment, socio-cultural factors, problems of communication, and the «trainer's» own limitation pose difficulties in the training of foreign country nationals.
8) The need for more research on the application of known and sound social science principles to health education as well as the need for more tools in research.

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EDUCAÇÃO SANITÁRIA EM SAÚDE ESCOLAR

DR.ª DEOLINDA DA COSTA MARTINS

Chefe da Repartição de Saúde Escolar e Educação Física
em Moçambique

Para melhor orientação do programa resolveu-se dividi-lo em três partes, dando-lhe a duração de três anos: 1.º ano, informação; 2.º ano, prospecção de hábitos alimentares; 3.º ano, aplicação da informação adquirida.

I PARTE (1956-57)

Tendo em vista a criação de uma «consciência de nutrição» entre as pessoas e organismos interessados na saúde das crianças em idade escolar — Serviços de Saúde, Serviços de Instrução, Comissão de Nutrição, Mocidade Portuguesa, Assistência Pública, pais, professores, professoras-visitadoras e o público em geral —, organizou-se um *programa activo* de informação e propaganda, que foi realizado pelos alunos do Liceu Salazar e do ensino primário oficial.

Com esse fim foi criada uma Comissão de Nutrição, que tinha como presidente a autora, ao tempo médica escolar das 2.ª e 3.ª secções do Liceu Salazar.

Essa Comissão levou a efeito:

1) Experiências dietéticas em ratinhos;
2) Trabalhos práticos de carácter técnico;
3) Palestras pela rádio e artigos nos jornais locais;
4) Exibição de filmes para os professores e alunos dos vários estabelecimentos de ensino;

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 569-578, 1959

5) Colóquios sobre nutrição para todos os professores de todo o ensino primário da cidade de Lourenço Marques;
6) A dramatização do tema «Bons dentes para os seus filhos» por um grupo de alunos ensaiados pelos seus professores;
7) O estudo das indústrias de alimentação da província;
8) O estudo da curva normal de crescimento dos alunos e alunas do Liceu Salazar — trabalho publicado em separata do *Boletim da Sociedade de Estudos;*
9) Prospecção de hábitos alimentares em áreas urbanas, suburbanas e rurais;
10) Estudo das tabelas existentes para alimentação dos soldados e dos trabalhadores do serviço dos organismos oficiais e particulares.
11) Execução de cartazes, gráficos e panfletos para uma exposição pública, que se realizou no átrio do Liceu Salazar, com o resultado das actividades anteriores e informação de base.

Ilustram estas actividades 18 fotografias enviadas com o trabalho.

II PARTE (1957-58)

Neste segundo ano concentraram-se as actividades na prospecção dos hábitos alimentares dos alunos de todos os estabelecimentos de *ensino oficial* da cidade de Lourenço Marques — primário e secundário: liceal e técnico —, com uma população escolar de 6856 alunos, assim distribuídos:

Ensino primário oficial

Europeus	48 %
Amarelos	2 %
Indianos	21 %
Mistos	19 %
Africanos	6 %

Ensino secundário: comercial e técnico

Europeus	64 %
Amarelos	1 %
Indianos	11 %
Mistos	12 %
Africanos	5 %

Foram excluídas as escolas rudimentares por não dependerem desta Repartição.

A mostragem foi de 6,5 % e a orientação e planejamento desta prospecção foi a seguida nos anos de 1954-55 nas escolas do condado de Santa Clara, do estado da Califórnia, E. U. A., pelos Serviços de Nutrição do Departamento de Saúde Pública do mesmo estado.

A prospecção teve por *fim:*

1) Obter informação sobre as práticas alimentares das crianças em idade escolar, relacionando-as com a raça, cultura, meio social e económico;
2) Utilizar esta informação como guia nas práticas alimentares futuras e no conteúdo de ensinamentos a introduzir nos programas de higiene;
3) Estimular o interesse do público em geral pelo desenvolvimento de um programa de nutrição;
4) Organizar uma prospecção-piloto que servisse de base para uma prospecção geral em toda a província, compreendendo todos os graus de ensino.

A prospecção foi levada a efeito pelos professores que foram iniciados na *técnica* de preenchimento das fichas por guias-referências (ver anexo).

Após experiências individuais e sessões de grupos de discussão, chegou-se à conclusão de que a recolha dos dados, para preenchimento das fichas, devia variar conforme os diferentes graus de ensino.

Assim, nas escolas primárias decidiu-se:

a) Pedir às crianças que na primeira manhã da prospecção fizessem um exercício de redacção sobre os alimentos ingeridos;

b) Discutir na classe, no dia seguinte, após o segundo exercício de redacção sobre o tema, os alimentos tomados, sua qualidade e confecção;
c) Sugerir às crianças que trouxessem de casa os alimentos estranhos;
d) Preencher as fichas só após estes grupos de discussão.

No ensino secundário:

a) Seguir o método anterior, o exercício de redacção, ou o preenchimento individual das fichas conforme o grupo de idades;
b) Respeitar os complexos económico-sociais próprios desta idade, não discutindo na classe a quantidade e qualidade dos alimentos ingeridos;
c) Inquirir, individualmente, após o preenchimento das fichas, para correcção quanto possível aproximada da verdade.

Na escolha da mostragem também se seguiram métodos diferentes, conforme o grau de ensino.

Assim:

No ensino primário escolheu-se, entre as 2.ª, 3.ª e 4.ª classes, a que, nessa escola, melhor representasse o grupo económico-social do bairro.

No ensino secundário escolheram-se as classes representativas dos vários grupos de idade e na secção mista do Liceu Salazar os alunos do 7.º ano que tinham colaborado activamente no programa informativo de 1956-57.

Não houve neste grau de ensino a preocupação de escolher grupos económico-sociais, por estes estarem mais ou menos seleccionados pelos estabelecimentos de ensino.

A *classificação* foi feita por grupos de alimentos e a avaliação por número de serviços em fichas individuais.

Para isso seguiu-se o guia (Form-Nutr-174-175-176) da prospecção de hábitos alimentares feito pelo Departamento de Saúde Pública do Estado da Califórnia, já citado, e consultou-se o nutricionista dos Serviços de Saúde.

Os alimentos estranhos, tais como: elchi, maga, jetinete, ciró, dara, etc. — dos alunos hindus —, foram classificados pelos Serviços de Agricultura de Moçambique.

Os resumos das fichas e as percentagens encontradas levaram-nos às seguintes *conclusões:*

1) Os alunos indianos e mistos são os que apresentam maiores carências alimentares, tanto quantitativa como qualitativamente. Os primeiros por educação ou religião e os segundos por razões económicas e sociais.
2) Os alunos africanos que mantêm a sua cozinha nativa têm uma alimentação mais equilibrada que os mistos ou os africanos assimilados.

> Exemplificando:
>
> O prato de farinha pilada com o caril de amendoim, de camarão seco e de folhas verdes de abóbora, rama de feijão cafreal ou cacana (vegetal verde); os frutos tropicais, como a maçala, o cânhamo, etc., e as oleaginosas, como a mafurra, são desprezados pelos mistos, que, em compensação, não têm em geral possibilidades económicas para comprar vegetais verdes e amarelos exóticos, de preço mais elevado, que substituam os anteriores.
>
> A análise das percentagens das fichas dos africanos, no ensino secundário, vem reforçar esta conclusão.

3) Aos alunos hindus faltam-lhes as proteínas animais e, tanto a alguns destes como dos maometanos, os jejuns a que estão habituados permite estarem na escola apenas com duas chávenas de chá e algum pão de manhã e à noite.
4) O pequeno almoço não nos aparece como refeição omissa, porque considerámos como base o pequeno almoço europeu: café ou chá com ou sem leite e pão com manteiga ou compota.
5) O almoço servido às crianças das caixas escolares, com carácter beneficente, por um organismo particular, está

carenciado; devia preencher, pelo menos, metade das necessidades diárias.

6) O chocoleite (leite desnatado com cacau e açúcar) servido às crianças das caixas escolares é a única fonte que recebem deste alimento.

7) Em toda a população escolar há nítida deficiência e em algumas escolas completa ausência de *vegetais verdes e amarelos, ovos, frutas, batatas, leite e seus derivados.*

8) As percentagens variam com os grupos económico-sociais.

9) O pão é a alimentação de base da população escolar; este, não sendo integral, não enriquecido e de tipo de muito miolo e pouca crosta, não tem as qualidades indispensáveis para alimento de base. Não sendo, em geral, comido com carne ou queijo, nem acompanhado de leite, não traz melhoramento na dieta diária do consumo geral das proteínas.

10) Os pastéis, rebuçados e laranjadas não fazem parte do consumo diário, em geral por razões económicas.

11) O almoço frio fornecido, a título experimental, aos 115 alunos da Caixa Escolar da Escola João de Deus, numa percentagem por raças de:

Europeus	10 %
Mistos	65 %
Africanos	24 %

sendo destes 10 % de hindus e maometanos, e constituído por: ¼ de litro de leite, 2 sanduíches (sendo uma de carne, guarnecida com uma folha de alface ou rodelas de tomate, e outra com compota de papaia), um ovo cozido por semana, em substituição da sanduíche de carne, e 2 bananas, foi aceite por todas as crianças, independentemente de raça ou religião, o que nos levou à conclusão de que os pais maometanos ou hindus, desde que não seja em sua casa, aceitam a carne como alimento, mesmo que esta não seja morta com os seus ritos religiosos.

III PARTE (1958-59)

Planejar de modo a:

1) Prover a que sejam remediadas as carências apontadas que possam ter resolução imediata;
2) Fazer um estudo da aculturação e do meio económico-social no qual nos possamos basear para organizar um programa de educação sanitária para as escolas e público em geral;
3) Estudar os recursos oficiais e particulares;
4) Envolver neste programa o maior número de grupos de população em cooperação activa com a colaboração da imprensa e a da rádio, de modo a acordar a «consciência de nutrição» na população da província em geral e nas camadas dirigentes e responsáveis em particular.

IV PARTE

Anexos:

1 — Guia-referência para os professores;
2 — Modelos da ficha individual e da sinopse dos alimentos consumidos;
3 — Sumário das fichas preenchidas (duas) respeitantes ao ensino primário e secundário.

Ficha de alimentos consumidos

Em ______________ ______________
Dia da semana — Mês dia ano

Nome :
Idade ☐ Masc. ☐ Fem.

Endereço : Lourenço Marques
Raça ☐ Afric. ☐ India. ☐ Mista ☐ Br. ☐ Amar.

Estabelecimento de ensino :	Ano	Turma

Escreva o nome do que comeu e do que bebeu		e quanto
Pequeno almoço		
Entre o pequeno almoço e o almoço		
Almoço (marque com uma cruz) ☐ Lanche trazido de casa ☐ Almoço em casa ☐ Almoço na escola ☐ Lanche comprado na leit. ou cantina ☐ Pensão ou restaurante		
Entre o almoço e o jantar		
Jantar		
Depois do jantar		

Não escreva aqui		Número
Alimentos	Número de serviços	Unidades totais
V. ou A.		
VC — Total		
Cit. 1		
T. 1/2		
C. 1/4		
S. V. 1/2		
Outras		
Bat.		
O. F.		
O. V.		
L. total		
L. 1		
Sor. 1/4		
C. ou A. total		
C. P. G. 1		
O. 1/2		
L. 1/2		
Q. 1/2		
A 1/4		
Sd. 1/4		
Cer. C.		
P. D. F.		
Ma.		
M. M. A.		
Pas.		
Reb.		
Refr.		
Chá e café		
Outra		

N M :
☐ L ☐ E. ☐ C. ☐ Ca. ☐ P. ☐ D.

Omitido :
☐ P. ☐ A. ☐ J.

Iniciais :

Sinopse das fichas dos alimentos consumidos

Lourenço Marques		Estabelecimento de ensino	Ano	Data

Número da ficha																					Total			
Indique o número de serviços																					0	Menos de 1	1	Mais de 1
V. ou A.																								
VC — Total																								
Cit. 1																								
T. 1/2																								
C 1/4																								
S. V. 1/2																								
Outras																								
Bat.																								
O. F.																								
O. V.																								
L. total																								
L 1																								
Sor. 1/4																								
C. ou A. total																								
C. P. G. 1																								
O. 1/2																								
L. 1/2																								
Q. 1/2																								
A. 1/4																								
S. 1/4																								
Cer. C.																								
P. D. F.																								
Ma.																								
M. M. A.																								
Pas.																								
Reb.																								
Refr.																								
Chá e café																								
Outra																								

	Iniciais																				L.	E.	C.	Ca.	P.	D.
Tipo de almoço																										

	Iniciais																				Pequeno almoço	Almoço	Jantar
Refeições omitidas																							

Detalhe para o total de leite acima de 1 copo:

	2	3	4	5	6	7	8	Acima de 8
Número de fichas								

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10. Panfletos, fotografias, cartazes e mapas fornecidos por:
    - *a*) Escola de Saúde Pública da Universidade da Califórnia;
    - *b*) Departamento da Saúde Pública do Estado de Oklahoma;
    - *c*) Serviços de Nutrição da África Equatorial Francesa;
    - *d*) Serviços de Educação Sanitária do Congo Belga;
    - *e*) F. A. O., Roma;
    - *f*) Metropolitan Life Insurance of New York;
    - *g*) General-Mills, Minnesota;
    - *h*) National Dairy Council, Chicago.

Guia para os professores na condução da prospecção de hábitos alimentares

Estude este guia cuidadosamente antes de distribuir a ficha aos alunos

1) Assegure às crianças que não serão classificadas nem criticadas pelo que escreverem na ficha de prospecção.

2) Estimule as crianças lembrando-lhes «ser um compromisso de honra» dizerem a verdade e que não devem copiar ou comparar a ficha do companheiro.

3) Não falar no trabalho que se vai fazer, a não ser no próprio dia em que se começa o inquérito.

4) A ficha destina-se a registar tudo o que se puser na boca e engolir durante 24 horas.

5) Deve dar-se bastante tempo às crianças para recordarem e registarem o que comeram e o que beberam.

6) Siga estas directrizes:

a) De manhã peça aos alunos para escreverem tudo o que comeram e beberam ao jantar, depois do jantar, ao pequeno almoço e ao lanche dessa manhã;
b) No dia seguinte peça para os alunos escreverem o que comeram e beberam ao almoço e ao lanche da tarde.

7) Não se devem dar exemplos de alimentos, como laranjas, flocos de aveia, etc., quando se dão instruções. As crianças podem pensar que julgamos que eles deviam ter comido esses alimentos; também os pode confundir a ideia do que gostam com o que comem.

8) Enquanto os vigiar no preenchimento das fichas, veja se eles registam a espécie de alimentos e o número de serviços (número de vezes que se serviram) de cada alimento.

9) Como devem ser registados os alimentos:

a) *Bebidas* — Espécie e número de copos de leite, cacau, sumos, coca-cola, etc.;
b) *Frutos e sumos* — Espécie e quantidade;
c) *Vegetais* — Espécie e quantidade;
d) *Ovos* — Quantos;
e) *Carne, peixe, galinha e queijo* — Quantas vezes se serviu;
f) *Combinações de alimentos* (espécie de ingredientes)[1]:
  Saladas — Como eram compostas;
  Sanduíches — O que continham.
  Sopas — *Canja*. Tinha carnes, vegetais, leite?
  Guisado — Tinha vegetais? E de que espécie?
  Torta — De que espécie?
  Pudins — De que espécie?

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[1] Muito importante.

CAUSAS DA CARÊNCIA PROTEICA NUMA REGIÃO TROPICAL: PERNAMBUCO, BRASIL

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O estado de Pernambuco situa-se em faixa paralela ao equador, a 8° de latitude sul, com a extensão de mais de 800 km e uma superfície de 99 000 km^2.

Vasconcelos Sobrinho (41) divide o estado, de acordo com o solo, o clima e a flora, em zona da mata e zona da caatinga. A zona da mata é de solo argiloso, profundo, húmido, de humidade relativa elevada, de índice pluviométrico favorável. Subdivide-se em uma subzona marítima costeira e outra continental. É a zona da mata utilizada na cultura da cana-de-açúcar. A segunda zona, a da caatinga, é, segundo o autor, de solo escasso e inexistente, atmosfera seca, baixo regime pluviométrico e pouca água no subsolo. A vegetação é xerófila. Esta zona é subdividida em zona do agreste e zona do sertão. A zona do agreste possui vegetação arbórea e pràticamente não tem gramíneas; a vegetação é quase sempre constituída de leguminosas e é uma área reservada à produção de cereais e à criação. Nela cultiva-se o feijão, o milho, o café, os frutos, o algodão, a mandioca, os tomates e a goiaba, oferecendo ainda possibilidades para a cultura do trigo. É zona de criação de bovinos, caprinos e aves. A zona do sertão caracteriza-se por solo sem humo, porém fértil e amplamente arejado. Não possui adequada protecção vegetal e é utilizada para criação de caprinos, asininos, aves e bovinos. A agricultura consiste no cultivo do algodão, do milho e do feijão. A zona do sertão é geralmente de clima frio e seco e sujeita às secas periódicas.

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A posição geográfica do estado de Pernambuco e, especialmente, da cidade do Recife — cujo porto está próximo da Europa — justifica a posição do estado na região do Nordeste brasileiro e mesmo no Brasil. Os estudos económico-sociais revelam que a cidade do Recife representa um centro de distribuição para outros estados e um centro industrial de significação apreciável, como se pode deduzir do quadro n.º 1 (2).

O problema da proteína na nutrição humana é um dos mais importantes e vem despertando o maior interesse em todo o mundo, quer se considere do ponto de vista nutritivo, quer do económico-social. São as propriedades da proteína como elemento específico do protoplasma, e conferindo-lhe diversas funções de fundamental importância, que lhe dão esse relevo. É do conhecimento geral que o estado de saúde de um povo e de grupos sociais depende principalmente da qualidade e da quantidade das proteínas ingeridas.

Diz Howe (31) que, como cada vez mais interesses nutricionais estão sendo conduzidos nas áreas mais subdesenvolvidas do mundo, se está tornando cada vez mais claro que a falta de um nutriente mais significativa é a de proteína, especialmente de alto valor biológico.

Devido à maior dificuldade de produção dos alimentos proteicos de valor biológico elevado, tornando-os mais caros do que os hidratos de carbono e mesmo as gorduras, as carências proteicas relacionam-se directamente com as condições do estado económico-social de um povo ou de um agrupamento humano.

Nos países de solos ricos, de facilidade de vias naturais de comunicação, de vegetação exuberante, nos quais a vida animal é fácil, e nas regiões onde a pesca é bem desenvolvida, a média de vida é, de um modo geral, elevada e os coeficientes de mortalidade — especialmente de mortalidade infantil, mortalidade por tuberculose e por parasitoses intestinais — são baixos. Por outro lado, os povos que vivem nos solos pobres ou esgotados, solos predominantemente ácidos e pouco férteis, onde a vegetação é pobre e a agricultura de subsistência é precária e de baixo rendimento económico, as carências alimentares, e especialmente a proteica, assumem grandes proporções, baixando a média de vida e aumentando os coeficientes de mortalidade (agravando as manifestações clínicas de verminoses, esq.).

É indiscutível o papel do solo no desenvolvimento e na estabilidade das civilizações. Os solos das regiões tropicais são, de um modo geral, ácidos e menos férteis do que os solos alcalinos, que se encontram, de preferência, nas regiões frias e temperadas, e por isso as civilizações dos trópicos se desenvolvem mais lentamente, enfrentam maiores obstáculos naturais e estão sujeitas a oscilações, declínios e decadências, principalmente quando os solos são empobrecidos pela destruição da cortina vegetal.

A produção de alimentos e sua oferta às populações em quantidades razoáveis é o factor principal da saúde de um povo. Se não existem em quantidades suficientes, a propaganda em favor da boa alimentação torna-se ineficaz. Os hábitos alimentares estão também ligados e em parte dependentes dos alimentos mais fàcilmente ao alcance. Há um paralelismo estreito entre o baixo nível económico, a nutrição e a saúde do povo. Nos países de baixo nível económico, de produção difícil e custo elevado dos géneros alimentícios apresentam-se todas as modalidades de carências — expressões da subnutrição e de fome crónica. E quando a deficiência recai na proteína animal, a repercussão é muito maior.

Diversos factores contribuem para que entre os povos varie o consumo proteico e os limites fisiológicos não estão ainda bem definidos. Uma quota de proteínas considerada óptima numa região que se apresenta com determinadas condições de temperatura, humidade relativa do ar atmosférico e onde predominam certos hábitos alimentares torna-se inadequada noutras, onde a temperatura é mais baixa, o clima é mais seco e os hábitos alimentares são diferentes.

O facto é que desconhecemos ainda o mínimo e a média proteica fisiológica na nutrição humana, e por isso não podemos classificar como deficientes dietas que revelam quotas de proteína inferiores às estabelecidas nas bases actuais, sem uma grande margem de segurança. Os que têm estudado o problema surpreendem-se com a insignificância de ingestão, por períodos longos, de proteínas animais ou mesmo de proteínas totais que teòricamente deviam produzir graves consequências ou seriam mesmo incompatíveis com a vida, sem que, em realidade, tenham grande repercussão. Estes factos põem em destaque a necessidade de conhecermos melhor a fisiologia do homem dos trópicos, para que lhe não sejam aplicadas normas benéficas apenas para o homem dos climas frios e temperados. Mesmo nos trabalhos experimentais não é possível ainda fixar a dose óptima de proteínas.

As divergências entre grandes autoridades sobre a média de proteína e o mínimo fisiológico são muito nítidas. Assim é que Voit estabeleceu uma média de proteína *per capita* de 118 g diários. Chittenden admitiu que uma quota de 44 g a 53 g diários permitia boa saúde e manteve seu equilíbrio proteico com apenas 36 g. Sieven obteve equilíbrio proteico com 25 g e McLister atribuiu a eficiência do soldado norte-americano na última guerra à quota de 180 g diários. Para Shermann, a quota de 44,4 g diários é suficiente, o que é confirmado por Rose. O Comité de Nutrição do Conselho de Pesquisas dos Estados Unidos estabelece 1 g/kg de peso corporal e o Ministério Britânico de Saúde aconselha 50 g de proteína de 1.ª classe (leite, ovos, queijo, carne e peixe). Segundo Houssay (18), o camponês argentino comia de 2 kg a 3 kg de carne por dia, o que dá uma quantidade muito elevada de proteína. Aykroyd (1) admite 2 g/100 Cal.

Segundo Terroine (44), os dados mais recentes sobre o mínimo necessário para produzir o balanço nitrogenado no adulto são os de Stare e colaboradores, publicados em 1946, os quais estão de acordo com os de Rubner na primeira década do século. Todos concordam que, utilizando proteínas de elevado valor biológico, 25 g por dia são suficientes para manter o balanço nitrogenado no adulto, se as necessidades calóricas forem adequadamente cobertas.

Waterlow (45) diz que o critério sobre o balanço nitrogenado de Terroine para o mínimo fisiológico em adultos é inadequado, porquanto uma pessoa pode estar em balanço nitrogenado com ingestão elevada ou baixa de proteínas.

Para as crianças, e até à puberdade, a quota aconselhada é de 2-3 g/kg/dia, devendo ser metade de proteína animal. Foi demonstrado na América Central e na África que, com 2 g ou 3 g de proteína vegetal por dia, o crescimento tende a seguir a curva normal, mas é retardado.

Diz Aykroyd (1) que há um grande número de informações de diferentes partes do mundo sobre as dietas nas quais os seres humanos podem crescer e viver razoàvelmente ainda com dietas relativamente sem proteínas animais, tais como as do leite.

O Comité da FAO sobre nutrição (25) sugere 0,35 g/kg de peso corporal como a necessidade mínima de proteínas por dia para o adulto.

É evidente a interferência de factores diversos para a média proteica. O clima tem grande importância, porquanto os climas frios e temperados estimulam a termogénese, a qual se faz, em grande parte, à custa das proteínas. Nos climas quentes o metabolismo basal é mais baixo e a termogénese reduz-se como meio de defesa contra a temperatura ambiente elevada.

Outros factores interferem no metabolismo proteico e na fixação do nitrogénio. É bem conhecido o papel de certos elementos minerais existentes no solo na fixação do nitrogénio pelas plantas. Foram já bem estudados na nutrição animal esses elementos minerais, a vitamina B_{12}, a vitamina B_6 e a constituição química do hidrato de carbono no metabolismo proteico e na fixação do nitrogénio. Diversos trabalhos revelam como o amido pode agir, modificando a utilização das proteínas e a fixação do nitrogénio. Assim, Harper e Katayama (30) demonstraram que, com dietas de 9 % de caseína, o crescimento de ratos era melhor com o amido ou dextrina do que com a sacarose. O amido do milho revelou melhor crescimento. Com dietas de 18 % de caseína não observaram diferenças nítidas. Para os autores as diferenças do crescimento não decorrem do sabor nem tão-pouco da influência da proteína ainda presente no amido do milho, mas sim ao facto de o amido do milho reduzir a velocidade do bolo alimentar no tubo digestivo, proporcionando mais tempo para o aproveitamento da proteína. Admitem também que a dextrina influencia benèficamente na utilização de aminácidos essenciais. Esses autores verificaram que a dextrina proporcionava melhor crescimento do que a sacarose numa dieta na qual a proteína era dada como caseína, albumina do ovo ou uma mistura de aminácidos com a constituição de caseína.

Madelyn Womack e colaboradores (46) observaram que ratos que receberam dextrina do milho como hidratos de carbono tinham equilíbrio nitrogenado, com quotas de aminácidos essenciais que produziam balanço nitrogenado negativo quando o hidrato de carbono era a sacarose. Substituindo a sacarose por dextrina da batata, observaram melhoria significativa do balanço nitrogenado nas rações com quotas baixas de aminácidos, mas não houve diferença com as de quotas elevadas. Os autores admitem, para interpretar o fenómeno, as seguintes possibilidades: 1.°, a dextrina pode fornecer pequenas quantidades adicionais de aminácidos essenciais ou não essenciais; 2.°, o aumento moderado calórico da dieta à base de dextrina pode ser benéfico ao animal; 3.°, há possibilidade de um tipo diferente de flora bacteriana em presença da dextrina modificando a utilização dos aminácidos; 4.°, diferenças do estado físico da dextrina e da sacarose podem modificar a eliminação do conteúdo intestinal, permitindo variar a absorção dos aminácidos.

Estudámos (19, 20, 43) experimentalmente as diferenças entre o amido da mandioca, o do milho e o da sacarose na fixação e utilização do nitrogénio em ratos brancos. Empregámos o teste da eficiência proteica de Osborne-Mendel-Ferry para estudar a curva de crescimento e ponderal e, para verificar a repercussão hepática das dietas em observação, doseámos os lipidos pelo método de extracção com o éter de petróleo ao Soxhlet e doseámos o nitrogénio pelo método de Pfluger. Empregámos ainda os métodos histoquímicos para dosagem dos lipidos hepáticos (o método de Sudan-Black) e do glicogénio hepático (o ácido periódico de Schiff). Empregámos dietas com quotas proteicas de 8 e 16 % (caseína) e lipidos nos níveis de 5 e 15 %. Cada grupo era formado de 10 ratos (5 machos e 5 fêmeas). Com a dieta de 8 % de caseína observámos infiltração gordurosa no fígado e teor baixo do glicogénio hepático com as dietas a 5 e a 15 % de gordura, em ambos os sexos e com qualquer dos hidratos de carbono (sacarose ou amido da mandioca), em comparação com o amido do milho. Com a dieta de 16 % de caseína não observámos infiltração gorda nos animais cujo hidrato de carbono era o amido do milho. Mas observámos infiltração discreta com os que recebiam sacarose e muito intensa com os que ingeriam o amido da mandioca. O teor do glicogénio hepático revelou-se na ordem inversa de infiltração gorda e o teste da eficiência proteica não mostrou diferenças significativas.

Esses factos demonstram como um simples factor — a qualidade do hidrato de carbono — pode influir na utilização e fixação do nitrogénio e, em consequência disso, na média fisiológica proteica. Como a mandioca contribui com grande parte da quota de hidratos de carbono na região em estudo e em outras regiões tropicais, é possível que esse factor importe na utilização das proteínas e seja responsável, pelo menos em parte, por modificações na utilização proteica.

É preciso um conhecimento mais preciso do valor biológico das proteínas mais utilizadas em determinada região para que realmente seja conhecido até onde elas podem interferir nas manifestações de carências proteicas. Por isso é que temos estudado algumas proteínas de origem vegetal que servem de base à quota proteica na região em estudo.

A importância dos feijões na campanha em favor da melhor quota proteica foi defendida na publicação da FAO *Síndrome Policarencial Infantil (Kwashiorkor) and its Prevention in Central America* (24).

Os feijões mais utilizados em Pernambuco são o mulatinho *(Phaseolus vulgaris)*, principalmente na região da mata, e o macassa *(Vigna sinensis)*, em grande parte do agreste.

Realizámos em nosso Instituto (13, 15, 17, 37) 4 trabalhos experimentais com o feijão mulatinho e verificámos que era inferior ao feijão macassa. Num trabalho utilizando o teste da eficiência proteica (curva ponderal) observámos a eficiência proteica do feijão macassa, ligeiramente inferior à da caseína: 88,92 % (16). Em outro trabalho (22) observámos em ratos brancos, tomando como padrão a caseína, o coeficiente de digestibilidade e o valor biológico das proteínas desse feijão, com a quota de 16 % de proteínas. Constatámos um valor biológico de 82,5 ± 1,05, muito aproximado do da caseína, que é 82,8 ± 1,13. O coeficiente de digestibilidade do macassa foi 88,2 ± 1,01 e o da caseína 95,3 ± 0,35. Também em nosso Instituto, A. G. de Matos Jr. (36) fez a análise cromatográfica do feijão macassa.

O feijão macassa é largamente utilizado na Baía e as baianas, tìpicamente vestidas, preparam o acarajé, que é feijão macassa com azeite de dendê e um pouco de pimenta; é um prato que se está irradiando em outras áreas do Brasil. Assim, nas ruas do Rio de Janeiro e no Recife já se encontram as baianas preparando o acarajé.

No laboratório de Mário Taveira foram analisadas 50 variedades de feijões utilizados no Brasil, tendo sido determinado o teor de aminácidos em algumas dessas variedades (3, 4, 5).

Hélio de Sousa Luz e Emília Pechnick (33) estudaram o feijão preto.

Ainda em nosso serviço, foi estudada a associação alimentar feijão mulatinho + xarque + farinha de mandioca (42) e o valor biológico, o coeficiente de digestibilidade e a eficiência proteica do caroço de jaca (35).

No campo das leguminosas foi objecto de nossos estudos a mucunã vermelha, esporàdicamente utilizada na região seca dos sertões no período das grandes secas, e concluímos pela inferioridade do valor biológico das suas proteínas, além da acentuada toxidez, obrigando a lavar a farinha de mucunã de 7 a 9 vezes (10, 12). Nossos trabalhos divergem dos de Josué de Castro com essa leguminosa acerca do valor nutritivo e da toxidez (6, 7, 8).

No prosseguimento do estudo das proteínas verificámos o valor biológico do sururu *(Mitilus alagoensis)*, muito utilizado no estado de Alagoas, próximo a Pernambuco e também usado em menor proporção neste estado (34). Os autores observaram que o sururu tinha eficiência proteica idêntica à da caseína e coeficiente de digestibilidade elevado, embora inferior ao desta última. O valor plástico revelou-se idêntico ao da caseína e o biológico no mesmo plano do das proteínas das carnes e peixes. O sururu foi também estudado, quanto à sua composição química, por Rubem Siqueira, Emília Pechnick, Nabuco Lopes e O. Guernelli (40).

Marcionilo Lins e colaboradores (32), da Faculdade de Medicina da Universidade do Recife, estudaram a composição em aminácidos da ostra e do aruá. Ambos revelaram metionina e são muito utilizados pela população pobre do Recife.

O problema da proteína e do clima é amplamente discutido na tese de Rui Coutinho (23).

Os inquéritos sobre carências alimentares, visando especialmente as de proteína na região, são ainda de número reduzido, não permitindo conclusões definitivas. A carência proteica é porém evidente, de um modo geral, na região do Nordeste brasileiro e revela-se através do retardamento do crescimento, do grande número de indivíduos com edema, da diminuição da imunidade no grande número de afecções carenciais e da grande incidência de anemias acentuadas. É evidente que outros factores, associados à carência proteica, contribuem para as afecções hepáticas, para os edemas carenciais, etc. A carência proteica revela-se mais acentuada na região da monocultura da cana-de-açúcar (região da mata), onde a agricultura de subsistência foi muito sacrificada. Os inquéritos feitos revelam deficiência calórica oscilando entre 1536 e 2170 calorias e pobreza de alimentos protectores.

Foram, entre outros, publicados os seguintes trabalhos: J. de Castro, Recife, 1934; A. Freire e A. Carolino Gonçalves, Recife, 1938; R. Coutinho, Recife, 1938; O. Parahym, Salgueiro, Pernambuco, 1943; J. F. Lima, Recife, 1943; O. L. da Costa, Alagoas, 1953; A. V. de Melo, Recife, 1947; G. de O. Ponce, 1947; Francisco Montenegro, Paulista, Pernambuco, 1952; Marcos Suassuna, Recife, 1948: refere carência proteica em crianças da cidade do Recife (42). Nelson Chaves (9) observou, em inquérito alimentar entre famílias de operários de empresa de transporte urbano, carência de proteína animal. Francisco Montenegro (42), em inquéritos entre operários de indústrias têxteis, revelou acentuada deficiência calórica (1465 Cal.), porém, quotas regulares de proteínas (52,2 g) e baixa de gorduras (23,3 g); a quota de hidratos de carbono encontrada pelo autor foi de 292,1 g. José Nivaldo e Neise G. de Sousa (39) estudaram a hepatose visceral e o *kwashiorkor* e encontraram na região do agreste de Pernambuco, numa investigação de oito anos, apenas dois casos, nos quais havia a sintomatologia clássica, inclusive a despigmentação dos cabelos.

A carência proteica no Brasil foi também posta em evidência pelo Comité de Nutrição da FAO, segundo Waterlow e Vergara (26). Os autores encontraram a distrofia pluricarencial em algumas cidades, notadamente Recife, Belo Horizonte e Rio de Janeiro. Consideram que o síndrome encontrado era bàsicamente o mesmo que a doença conhecida como *kwashiorkor* na África e que em certas regiões do Brasil a desnutrição atinge talvez ⅓ das crianças.

EXPRESSÕES DA CARÊNCIA PROTEICA EM PERNAMBUCO.

A carência proteica manifesta-se por:

a) Média de vida muito baixa;
b) Mortalidade infantil muito elevada;

c) Grande incidência de doenças hepáticas, inclusive cirrose;
d) Anemias;
e) Retardamento do crescimento;
f) Edemas carenciais.

CAUSAS DA CARÊNCIA PROTEICA.

a) Predominância de proteínas de origem vegetal (feijão mulatinho) sobre as de origem animal.

É um fenómeno comum que ocorre em muitas partes do mundo, especialmente nos países tropicais, devido à deficiência de criação de gado bovino, caprino, suíno e de aves e à falta de organização da pesca até muito recentemente. Na região em estudo, especialmente na zona da mata, o consumo de leite, ovos, carne e peixe é muito baixo. E na cidade do Recife, onde mais de 50 % da população é de baixo nível económico, o consumo de proteína animal tem sido muito reduzido.

As grandes áreas húmidas da região da mata são absorvidas pela cultura da cana e a criação na zona seca do sertão (subzona da caatinga) é muito difícil em face da escassez de água e das secas periódicas. Na zona do agreste (subzona da caatinga) está também sujeita à seca e à restrição de água, embora em menores proporções. Por isso, a criação de bovinos, que teve certo esplendor no período colonial, deslocou-se para outras regiões do Brasil, como Minas Gerais e Brasil Central, e assume actualmente grandes proporções no Rio Grande do Sul. Na região da mata a escassez de carne e leite e seus derivados é muito mais acentuada do que na região subárida.

O consumo do peixe foi até há pouco tempo baixíssimo, em virtude de a pesca não estar bem organizada no estado e na região do Nordeste de um modo geral. Basta dizer que peixe de 3.ª ou 5.ª categoria era vendido como de 1.ª categoria no Recife, capital de Pernambuco e uma das principais cidades brasileiras, por preço três vezes superior ao da carne de vaca. Sòmente há pouco mais de um ano é que um grupo de pescadores japoneses iniciou a pesca em moldes económicos e a população pôde usufruir as vantagens da riqueza da costa do Nordeste brasileiro em peixe de 1.ª categoria.

Assim, o abastecimento da cidade do Recife e de diversas cidades do interior vem sendo feito com certa regularidade, sendo o quilograma do peixe vendido por quase metade do preço da carne de vaca. Isto vai certamente modificar muito o estado carencial e, com a intensificação da pesca, o problema da proteína animal no estado de Pernambuco estará em grande parte resolvido. Acreditamos que a pesca em larga escala contribuirá para modificar radicalmente o actual *status* de subnutrição proteica em que vive a maior parte da população do estado de Pernambuco e do Nordeste.

b) Outro factor da carência proteica na região em estudo é a grande extensão territorial do Brasil, com população ainda muito reduzida e grande deficiência dos meios de transporte. Há no Brasil ricas fontes de proteína animal, mas a distribuição não se faz com eficiência nas regiões distantes, onde a produção é ainda escassa devido à falta de transporte. O Brasil possui um dos maiores rebanhos bovinos do mundo, concentrado nos estados do Rio Grande do Sul e Minas Gerais e no Brasil Central, o que permite um excelente *per capita* de carne de vaca e de leite para o homem. O Rio Grande do Sul está ameaçado de perder grande parte da sua produção de leite em virtude da dificuldade do meio de transporte e há cidades do Nordeste brasileiro e de Pernambuco nas quais as crianças não conheciam leite. A situação vem melhorando porque o Fundo Internacional de Socorro à Infância (FISI) vem distribuindo o leite desnatado para atenuar a carência de proteína animal.

Com transporte e meios de industrialização e de conservação adequados e a intensificação da pesca é possível transformar uma situação verdadeiramente calamitosa numa perfeitamente razoável ou mesmo óptima em relação ao *per capita* de proteína animal. É preciso que se organizem e executem planos nos quais o assunto seja considerado nos seus diversos aspectos e sejam mobilizados nutricionistas, médicos, agrónomos, químicos e economistas.

c) *Solo e cortina vegetal.* — Estando o estado de Pernambuco, bem como o Nordeste brasileiro, numa área tropical, não podia fugir às contingências impostas pelo solo e pelo clima. É o solo um complexo biológico muito dinâmico e mais ou menos variado, do qual participam a terra, o mundo vegetal e animal, resíduos vegetais, grandes números de bactérias e outros seres vivos. Sua fertilidade depende da composição, dos materiais orgânicos e inorgânicos, entre eles os oligoelementos, da riqueza de germes, do pH, do humo, da sombra das árvores, mantendo o grau de humidade necessário às bactérias, da erosão pela água ou pelos ventos, etc. Os vegetais constituem a principal protecção do solo, proporcionando seus restos — folhas, galhos, frutos, etc. —, sombra e humidade, atraindo água para as profundidades, protegendo-o da erosão pela água e pelo vento e das inundações. Certos vegetais, como as leguminosas, contêm bactérias que retiram o nitrogénio do ar atmosférico. Por esses motivos, representa a cortina vegetal o intermediário entre o solo, de um lado, e a vida animal, de outro. Dela depende o ciclo do carbono e do nitrogénio. São os vegetais verdes que, por intermédio de seus pigmentos — clorofilas, carotenóides, ficobilinas —, armazenam energia solar e realizam, através de uma série de reacções complexas, a síntese dos monossacaridos, os quais vão dar posteriormente di e polissacaridos e gorduras. O nitrogénio do solo, procedente dos restos animais ou vegetais e do ar atmosférico, vai juntar-se à molécula do hidrato de carbono para a formação da molécula proteica vegetal. O emprego dos isótopos tornou possível conhecer a sequência das reacções e o conjunto dos produtos intermediários da fotossíntese. Discutem-se alguns aspectos químicos da fotossíntese, como sejam a sequência de alguns produtos intermediários, a origem do oxigénio e o papel do fósforo, mas a importância geral da reacção é indiscutível. Sabe-se que as algas têm grande poder fotossintético, 5 vezes aproximadamente superior ao dos vegetais que vivem à superfície do solo.

Se o solo é rico em elementos minerais, é de boa espessura, não foi muito prejudicado pelas erosões, não tem laterite e é alcalino, como acontece geralmente nas regiões frias, a vegetação é exuberante e mais rica em substâncias orgânicas e elementos minerais. Há uma grande margem de variação na composição orgânica dos vegetais paralelamente à composição do solo. A riqueza da cortina vegetal acarreta também a riqueza da vida animal, o que condiciona abundância de alimentos para o homem, quer de origem vegetal quer de origem animal, e um bom *per capita* de proteína animal e vegetal.

Se a cortina vegetal se desenvolve num solo pobre, com escassez de elementos minerais e de água, ela é também pobre, protege menos o solo e constitui uma fonte de alimentos de menor significação para os animais e para o homem. O ciclo solo-vegetal-animal-solo é, evidentemente, menos eficiente quando o ponto de partida é pobre do que quando é rico.

Se nesse solo pobre, de cortina vegetal reduzida e com menor área para a fotossíntese, as árvores são cortadas em larga escala para fornecimento de energia e fins domésticos, tudo se agrava: o ciclo do carbono é prejudicado, a área destinada à fotossíntese é ainda mais reduzida e a erosão encontra o campo aberto para arrastar a parte fértil para os rios e para os mares. A devastação da floresta é um dos meios mais adequados para empobrecer um solo, reduzir-lhe a fertilidade ou mesmo preparar um deserto. É justamente isto o que se tem feito em larga escala no Brasil, principalmente no Nordeste, incluindo o estado de Pernambuco. A necessidade de energia para as fábricas e para as estradas de ferro, apesar de haver grandes fontes de energia no país, contribui para o empobrecimento dos solos cultiváveis.

Foi esta, sem dúvida, a causa principal da carência proteica na região estudada, porquanto tornou difícil a vida animal e teve como consequência imediata a escassez de leite, de carnes, de ovos, etc.

O documentário a respeito dessa devastação permite verificar que ela foi alarmante e não se limitou a essa região do Brasil, tendo abrangido também outras regiões, como Minas Gerais, São Paulo e Paraná. Com as facilidades de energia, como sejam energia hidráulica, nuclear e solar, é possível que se detenha nas regiões tropicais a destruição de florestas em larga escala. Há já exemplos impressionantes, como o relatado por Pierre Gourrou (28, 29) com respeito à ilha de Madagáscar, com 580 000 km^2, dos quais 500 000 km^2 se converteram numa região laterítica estéril em consequência da destruição de florestas. Refere ainda o mesmo autor a lenda da fertilidade da ilha de Trindade. Um outro exemplo é o da decadência da civilização maia, discutida ainda, mas interpretada por alguns, como Pierre Gourrou e Sylvanus Morley (29), como consequência da devastação de florestas, roubando o humo do solo e empobrecendo-o.

O assunto da devastação das florestas e sua repercussão na nutrição foi objecto de trabalhos anteriormente realizados por nós (14).

Diversos outros autores trataram do assunto e, entre eles, Vasconcelos Sobrinho (41) diz que a destruição se estendeu às árvores frutíferas de uma maneira impressionante. Diz ele que só no Recife foram destruídos, nos últimos vinte anos (até 1949), 380 000 cajueiros, 190 000 mangueiras, 3000 abacateiros, 30 000 bananeiras e 70 000 coqueiros.

Acontece que o cajueiro é uma árvore de grande importância na nutrição humana, pois fornece o caju, muito rico em vitamina C, e a castanha, que é fonte de proteína de boa qualidade e rica em gorduras. A castanha desempenhou um papel muito importante na nutrição do Nordestino, muito especialmente nas zonas de praias, onde era largamente usada torrada ou sob a forma de farinha. Foi amplamente empregada em Pernambuco e em outros estados na preparação de bolos e de pratos, como o vatapá e o caruru. Mauro Mota (38), num livro interessante, *O Cajueiro Nordestino,* salienta a importância do caju e da castanha sob vários aspectos e critica a devastação dos cajueiros, que vem sendo feita em larga escala.

d) Interferência de outros componentes do regime.

É possível que o regime de predominância vegetariana contribua para a deficiência de vitamina B_{12}, o que certamente restringe a síntese de proteínas no organismo e a fixação do nitrogénio.

É também muito provável que o amido da mandioca, por nós estudado e largamente utilizado no estado de Pernambuco e no Nordeste brasileiro de um modo geral, reduza a eficiência proteica e o valor biológico.

Todas essas causas tomadas em conjunto é que determinam a carência proteica actualmente dominante no estado de Pernambuco e em outras áreas do Brasil.

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THE ROLE AND TASK OF THE MEDICAL EXPERT CONSULTANT IN INTERNATIONNALY SPONSORED HEALTH PROGRAMMES

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> Where's the voice, however soft,
> One would hear so very oft?
>
> JOHN KEATS

In nearly all Public Health Campaigns the Medical Expert Consultant assumes an integral position as Chief Medical Adviser of a Health Control Programme, actively planned and developped for a specific purpose, and length of time.

To satisfy requests from Health Agencies of National Government especially from those in so-called «Underdeveloped Countries», the United Nations World Health Organization (WHO) and the United Nations Childrens Emergency Fund (UNICEF) have, and are choosing Medical Expert Consultants, usually from another country where they have attained the recognized stature in their respective speciality. In Resolution 222. A (IX) of the Economic and Social Council of the United Nations passed in August 1949 it is stipulated:

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STANDARDS OF WORK AND PERSONNEL

1. The highest professional competence should be maintained in all services undertaken by the participating organizations in rendering technical assistance to requesting countries.
2. Experts should be chosen not only for their technical competence, but also for their sympathetic understanding of the cultural backgrounds and specific needs of the countries to be assisted and for their capacity to adapt methods of work to local conditions, social and material.
3. Adequate preparation of experts should be provided before assignments are undertaken; such preparation should be designed to give understanding of the broad objectives of the common effort and to encourage open-mindedness and adaptability.
4. Experts and groups of experts visiting a country should not engage in political, commercial or any activities other than those for which they are sent. The scope of their duties should be strictly defined in each case by agreement between the country requesting assistance and the organizations providing assistance.
5. Even when allocations are committed, projects should not be commenced unless properly qualified experts and assistants have been secured and trained.

PARTICIPATION OF REQUESTING GOVERNMENTS

2 To give full and prompt consideration to the technical advice they receive as a result of their co-operation with the participating organizations in response to the requests they have initiated…

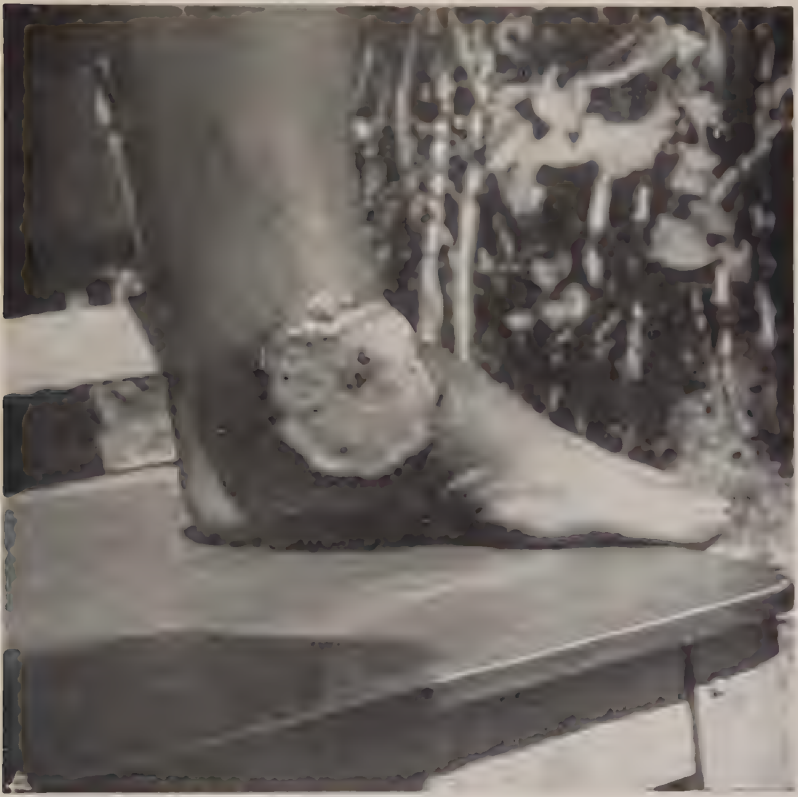
In the respective «Plans of Operations» (PLANOPS) of a specific Health «Campaign», «Programme», or «Project», respectively, as these terms are sometimes interchangeably and indiscriminately applied, usually merely a general over-all direction are enumerated pertaining to the presumed functions and activities of the Medical Expert Consultant in line with the stated general policies and objectives of the project to which he has been appointed, or assigned. Particular details, however, are nearly never determined, and, unless he has gained experience from former similar assignments, he must steer his own course. When I had been appointed as Chief Medical Adviser of the Treponematoses Control Project in Indonesia, the PLANOPS merely stipulated:

1. He will act as adviser to the Ministry.
   He will assist the Director, as requested, to help arrange for the evaluation of the work of the program, the details of its execution, plan the projects of the research team and coordinate the work generally.
2. He will prepare and forward to the Regional Director, WHO, with copies to UNICEF offices, monthly and yearly reports covering all operations of the teams, and these reports will include, as annexes, the reports of the Bacteriologist.

Inasmuch as such general direction of march is as broad as possible, such directives are necessarily subject to the «error and trial» method, but rather inevitable in the initial phase of any entirely new programme of any sort. Of course, with the illuminating benefit of hindsight criticism becomes easy, and always cheap.

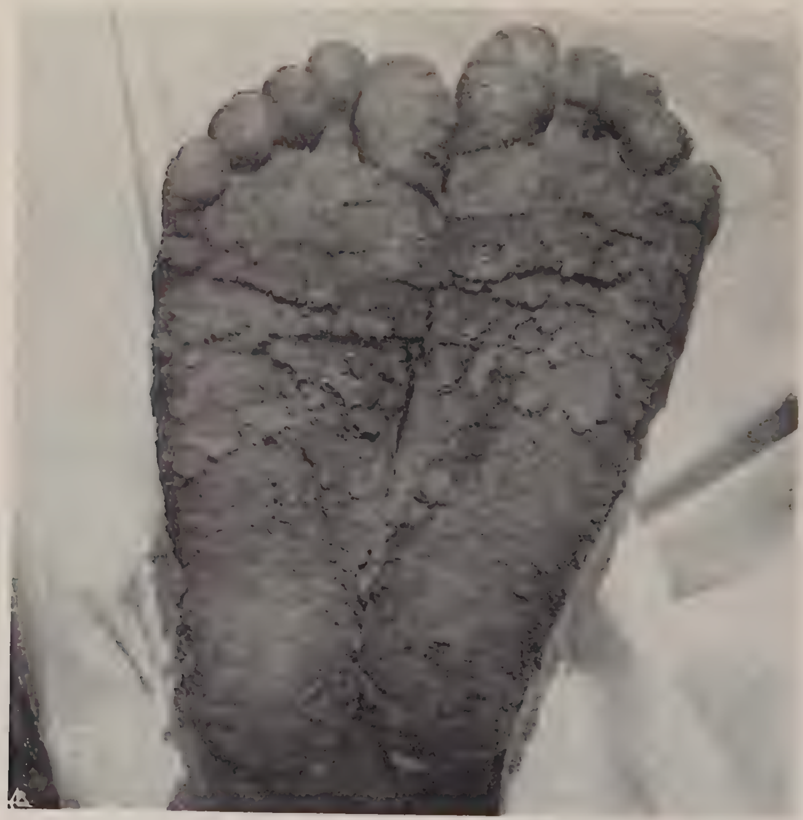
Similarily, my assignment 1937 by the U.S. Public Health Service to investigate the relationship of endemic Syphilis among the Arabs («Bejel», «Firjal», Abou-Laghif) to veneral Syphilis, and to Yaws, and again when asked by WHO in 1952 to assess the anti-Yaws project in the Philippine Islands, no specific detailed instructions were given, though in both instances, of course, my clinical and experimental experience of more than 20 years medical work in Tropical and foreign domains in general and South and Far Eastern Asia in particular stood to enhance dealing with the issues and problems, medical and sociological.

Whereas the academic standards for field personel should by no means be unnecessary high and, for them, more emphasis must be laid on whether they really possess command of their everyday business coupled with the spirit and endurance to carry it out under difficult and tough field conditions, the *conditio sine qua non* for any Medical Expert Consultant must be the sovereign command of the subject matter for which his work is destined. Though previous field work will undoubtedly enhance his know-how, and which he must have collected somewhere sometimes anyhow, adequate clinical and especially experimental experience ought to be a prerequisite, to my mind, lest the Consultant sadly lacks the profound command of essential knowledge pertaining to the technical and scientific problems and issues involved. It is rather immaterial whether purely clinical experience or policlinical (dispensary) work of sufficiently long duration



Initial "primary" Papilloma framboetica

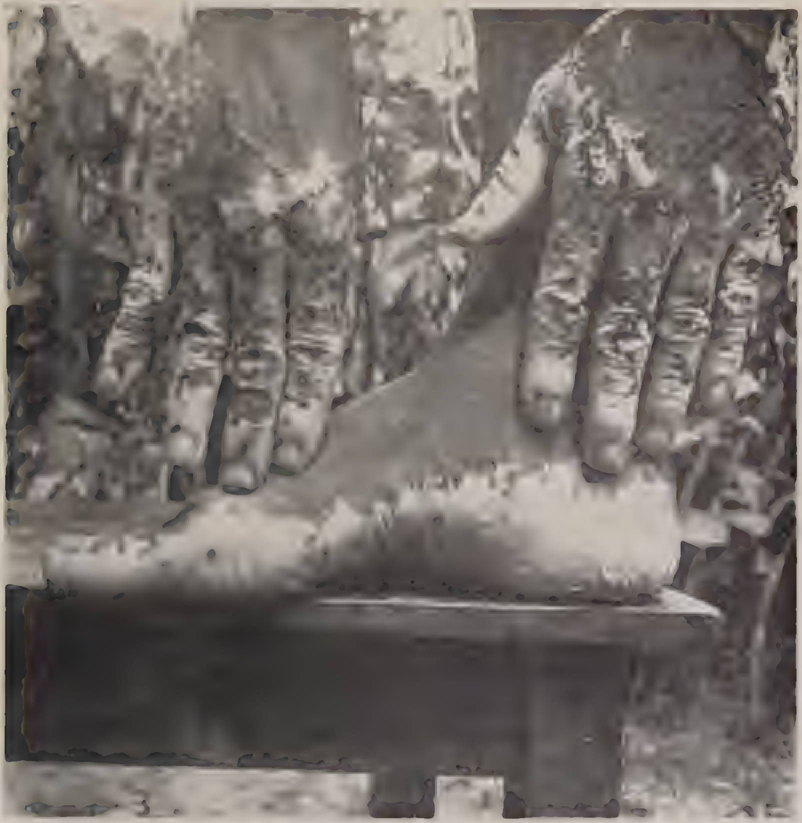
has been gained, but the main point is *adequate familiarity with the respective disease entity as running in the sick patient, not as described, and enumerated in text-books or mere classroom lectures.* To command authority, it seems also imperative that the Consultant is able to produce adequate evidence of previous experimental work supported by sound scientific publications, in his respective, realm in order to prove his established faculty. Unless he possesses such sovereignty regarding the subject, the National personnel and Authorities will soon detect his possible flaws, and his advice cannot be thoroughly competent and sound, both scientifically and technically,



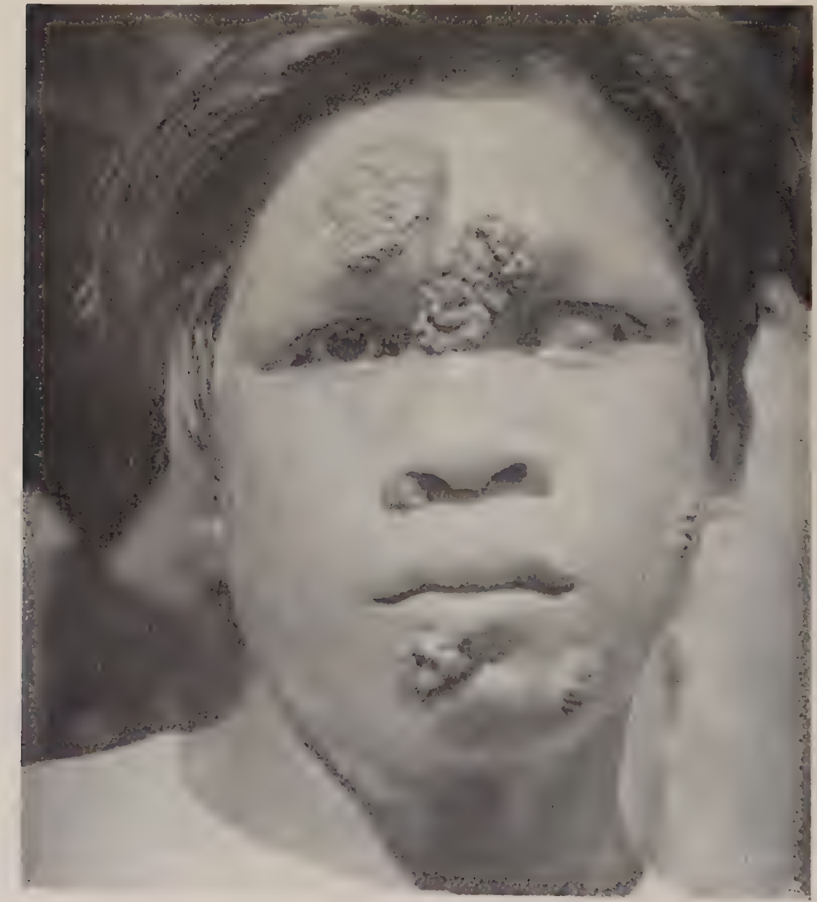
Keratodermia framboetica

and he might not for long remain totally disengaged from, nor unsoftened by possible larger considerations.

As for the rest and by no means to be taken lightly, the question of adaptibility appears to be of prime essence, especially so vis-a-vis the protean demands made upon any man working with people of often different background from his own. He must be free, above all, from any prejudices regarding race, color, religion or otherwise, and prepared to putting the principle in practice as getting to know people by associating with them officially as well as socially. Local customs vary greatly in different countries, and much as «perfectionism» in

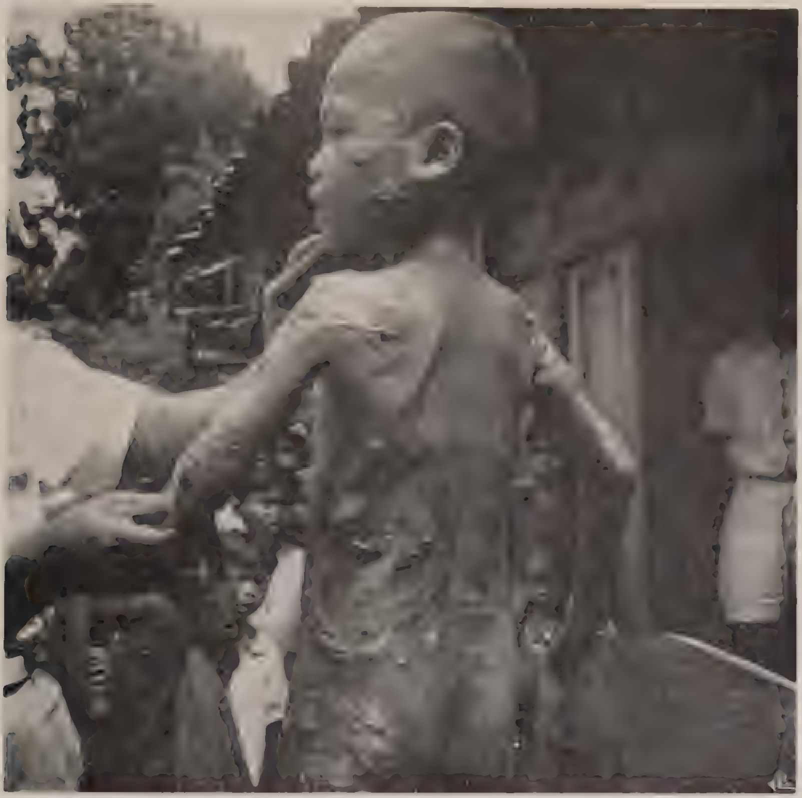


Keratodermia framboetica with Leucoderma ("Mbelung" of ZIEMANN) any line might be a goal to achieve in the interest of public health advancement in general and the objectives of any project in particular, the desire to change habits must be tempered with the final realization, yet at the same time appreciation of the importance of human relations and understanding in international mutual work. In selecting an Expert, consideration shoud be exercised regarding his own habits and «life pattern», and in this sex requirements should not be neglected.



Polypapillomata framboetica

The Medical Consultant should remain long enough on the job to accomplish the task for which he had been selected. This ought to be a minimum of 2 years. I would plead that provision is made for him to spend at least one month prior to his final assignment at an identical project in the field as on-the-job-training in the same, or in another country in order to get familiarized with exactly the same type of both advisory and field work required in that particular campaign. Another month, at least, he should be attached to any project in the *same* country of his assignment to get acquainted with mentality, usages and customs peculiar to that country.



Polypapillomata framboetica, coexistant with ulcerative late Yaws lesions

It is humanly understandable and common experience that a dedicated specialist is fond of his particular rôle and importance in the campaign, against which the desires and stated needs of the project as a whole must be properly balanced. Although it is not enough to represent merely one of the smooth and trusted Officials of one of the International Agencies «hierarchy», the spiritual Amok runner or the garrulous dissenter would both jeopardize the purposes for which he is sent, viz. assisting reasonably and efficiently in developping a health project. This does not imply, of course, to divorce himself entirely from that invigorating, priceless restlessness and self-dissatisfaction married to the sense of the deep importance of his mission to the welfare of mankind. Though his bounty is the sovereign command of the pertinent medical issues, his bane might often be the glaring problem of «getting along» with the limitations inherent in the National finiteness both regarding personnel and finance, and vis-a-vis which he must possess the necessary mental survival equipment. Whereas he should be constantly aware of the imperative condition that the entire control programme and its success rests on the full understanding and active cooperation of the people-at-large, the civil authorities and, last but not least, on the «saintly ferocity» of the medical teams, he must also preserve the distance necessary to command authority, and which ought to remain between master and apprentice, laying down the law without an eye to bias or partiality. This, of course, is quite different from any attitude of an allegedly «mind culturally sealed» or acting «like prima-donnas».

Surely, the pursuing of purely conscientious and strictly professional, scientific objectives falls more easily upon foreigners who possess assured life-long civil service status in their own home-countries, rather than upon officials or workers in U.N. Organizations or in the respective National Governments now-a-days; after all, the former do not have to run for office, or to keep on.

In considering the various aspects of the particular campaign of his special assignment, the Consultant should always keep in mind that all special health campaings such as against Malaria, Yaws, Tuberculosis, Trachoma a.o. should be ultimately integrated into the permanent health services of a community as functions of urban and rural health centres; hence the respective pertinent post-campaign activities must be co-ordinated with the other medical, and medico-social services.

To facilitate and enhance the time and fruitful labors of the expert Consultant arriving at his new duty station, the Resident Representative of his respective Organization can, if such exists, be of invaluable assistance in providing the immediate pertinent contacts with Government Agencies and their Officials. The same applies to contacts and cooperation with Chiefs and Experts of any other United Nations, or other foreign Expert Consultants where consultations and cooperation free from feelings of interference or possible jealousy are essential to enhancement of a pervasive confidence and sense of common cause bringing their varied actions into concert.

Inasmuch as practically all internationally sponsored public health programmes — with the exception of special emergency assistance in times of sudden disaster and pestilence — are singularly destined to initiate a definite objective to be integrated eventually into the National Health activities and to demonstrate to the respective national health personnel field procedures, etcetera, it is imperative both that the chosen national counterpart personnel is fully ready to assume its duties according to the stipulations laid down in the PLANOPS and that adequate clerical services and provision of able interpreters are fully provided from the time of the Consultant's arrival to avoid waste of time and frustration.

Pursuant to the old adjudgement «Cuius regio, eius religio», the portent of advice of the trusted representative of WHO and the prestige appurtenant upon his position as Expert Consultant should be augmented by the proviso that all requests and vouchers for foreign suplies and assistance emanating from the Field-Teams and/or the Program-Director should be honored by the foreign aid-agency (mostly UNICEF) only, if and when counter-signed, or at least specifically approved by the Special Consultant. This will materially attenuate unsound operational procedures, and possible inappropriate use of supplies for which, after all, foreign tax-payers foot the bill.

Finally, as a prophylaxis against red-tape, cumbersome protocoll and attenuating probable waste of time, in all PLANOPS should be stipulated for the Medical Expert Consultant «*To advise, demonstrate and train at all levels*» in order that he may contact Government Agencies at all levels, and assume stewardship demonstrating and advising anywhere, anytime and in any manner, he so chooses, in order that the objectives are enhanced, and not attenuated by otherwise retarding or inhibiting factors of various possibilities.

Yet, irrespective of all professional, scientific and practical knowledge and field experience supported by the necessary profound tact, dame luck must, last but not least, grace the Medical Expert Consultant to a large extent to steer clear on one hand, between the Scylla, of utter professional medical integrity and rapt fervour regarding the objectives he is charged to attain, but perhaps threatened with possibly incurring at the same time antagonistic feelings on the part of the alien Government and counterpart personnel, and the Charybdis, on the other hand, of harmoniously «getting-along» only too well and creating «good-fellowship» first and all, yet at the expense of, perhaps, unsound operational procedures, too much latitude vis-a-vis the strict code of professional duty and self-dedication to the job taken on, and, consequently, perhaps even disastrous failure to reach the ultimate targets of the prescribed Health objectives. For, the aims and labors must not be merely, or preponderantly measured in terms of propaganda and goodwill or perhaps, enchanting any special segment of local interest, but rather in terms of workability and envisaged achievements, of course.

FORMAÇÃO DE EDUCADORES SANITÁRIOS EM MOÇAMBIQUE

JOAQUIM FERREIRA DA SILVA

Entendendo-se por saúde um estado de bem-estar físico, mental e social do indivíduo, e não apenas a ausência de doença ou enfermidade, pode afirmar-se que a defesa sanitária dos indivíduos se firma cada vez mais em princípios de ordem social.

De facto, boa casa, correcta alimentação e vestuário suficiente são factores fundamentais de conservação de saúde e de defesa contra as doenças, numa afirmação preciosa da actual orientação da Medicina.

Pela apreciação destes conceitos se medem os problemas que constituem a tarefa do organismo, a cuja responsabilidade está confiado em Moçambique o nobilíssimo encargo de assegurar a saúde das populações, preservando-as da doença.

Os Serviços de Saúde exercem a sua actuação junto de uma população nativa de cerca de 6 milhões de habitantes, e exercem-na eficazmente.

E, apesar da notável modificação verificada já na saúde das populações por tal motivo, aparece-nos cada vez mais a necessidade de dominar toda a actividade dos Serviços pela prevenção da doença e educação sanitária — aspectos fundamentais da higiene e da medicina social —, cuja intervenção se impõe cada vez mais, para evitar o aparecimento de uma infinidade de causas malsãs, fruto de uma vida social mais complexa em perfeita relação com o progressivo desenvolvimento social das populações nativas, que do seu baixo nível de vida caminham precipitadamente para a melhoria da sua condição social e económica, em premente anseio de progresso.

E por isso sucede que, sempre que apreciamos a actuação dos Serviços de Saúde, em face desta palpável transformação social, ao fazer o balanço da obra realizada, depara-se-nos, na sua justa medida,

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o caminho andado e somos forçados à prática de tarefas mais amplas, mais complexas embora, que constituam o óptimo desejável, para que toda e qualquer acção sanitária se oriente no preciso caminho de em nada perdermos tempo.

E assim, tendo em conta os problemas da medicina hodierna, se verifica que é preciso mais que nunca olhar pela vida e saúde das populações, protegendo-as e preservando-as, sobretudo ao reconhecer que a saúde pública é o primordial elemento de força, progresso e felicidade de uma nação.

Por isso, no pleno conhecimento do meio em que os Serviços de Saúde exercem a sua acção, com base no próprio conceito de saúde, há que promover atitudes que visem o bem-estar físico, mental e social do indivíduo, numa relação constante com o nível económico da sua vida.

Só assim procedendo poderemos antepor à treva de uma teimosa rotina de exclusiva acção curativa o sol radioso da medicina preventiva, com vista a conseguir-se uma saúde vitoriosa em que se tenha por norma evitar a doença e dar ao corpo e ao espírito o máximo desenvolvimento normal.

Poderemos consegui-lo, certamente, pelo caminho da educação sanitária — expressão nova, baseada no espírito do velho aforismo que põe na escala dos valores úteis o prevenir acima do remediar — , utilizando a indispensável colaboração das populações.

Para o fazer há que criar hábitos de higiene nas populações, promovendo o saneamento das localidades, e promover com o maior interesse, antes do mais, a formação de educadores sanitários, pondo à disposição das populações pessoas competentes, zelosas e humanas, que conheçam e tenham atracção por problemas educativos e sociológicos.

Só assim se poderá iniciar uma larga tarefa, sem a realização da qual não haverá saúde, nem se prevenirá eficazmente a doença, nem haverá higiene e saúde bastantes à valorização económica do nativo. Tarefa eminentemente social esta da educação sanitária, que terá de contar necessàriamente com colaboradores competentes, para que se valorize a saúde das populações, do que dependerá, sem dúvida, o seu progresso social e material. Tarefa que terá de contar necessàriamente com a colaboração de todos os indivíduos.

A educação sanitária pretende fazer de cada indivíduo um colaborador consciente de medidas de higiene, profilaxia e terapêutica. Bastaria enunciar só por si este objectivo para se ter de concluir que seria impossível atingi-lo. Contentar-nos-emos, porém, em obter a adesão e o auxílio da maior soma de indivíduos, fazendo cooperar, sobretudo, aqueles que dentro da colectividade exercem, por dons naturais, por virtude de cargo ou por força da tradição, o papel de conselheiros e guias da população.

Até ao presente a educação sanitária tem sido obra de médicos. Em alguns países reconheceu-se, porém, a necessidade de preparar para essa missão uma unidade específica, com estudos especiais e uma profissão definida — o educador sanitário. Não é um profissional de arte de curar. É um profissional de educação e a sua acção exerce-se, sobretudo, junto das pessoas sãs.

O educador sanitário que não teve aceitação nem na estrutura clássica dos nossos Serviços de Saúde nem na inovação africana das formações móveis tem-na já no presente.

A evolução das técnicas de assistência e profilaxia regista nos últimos anos um aspecto essencial, traduzido no carácter dinâmico dos serviços médicos. A formação clássica recebia o doente. A formação actual vai buscá-lo, trata-o no local da sua residência, despista-o. Definiram-se claramente com a ambulância, a equipa móvel e o rastreio as características dinâmicas a que me refiro.

Foi dessa mobilidade da formação sanitária que nasceu em África a necessidade da propaganda e informação, que até esta data já conta de resto algumas realizações práticas. Falta dar agora mais um passo. Entregar a propaganda e informação a entidades especialmente preparadas e, ao mesmo tempo, ultrapassar a fase da informação, entrando no caminho da educação. Qual a diferença? Onde está a fronteira? A educação é sinónimo de mudança de hábitos. Educação sanitária é mudança de mentalidade e mudança de atitudes.

Tudo isto não pode, porém, ser passivo e não pode decorrer apenas como consequência da informação e propaganda.

É necessário que a população participe activamente na solução dos problemas de higiene, profilaxia e terapêutica para que se opere a mudança de atitudes e, portanto, se inicie o capítulo da «educação sanitária».

Aberto assim este novo campo de trabalho, falta em Moçambique conhecer o terreno onde ele terá de executar-se de uma forma sistematizada, examinando em conjunto todas as experiências realizadas e tirar delas normas para a acção futura.

A primeira lacuna que nos aparece, porém, é a falta de um estudo sociológico, constando de um inquérito sistematizado sobre a demografia, psicologia, hábitos alimentares, higiene geral e demais problemas inerentes à assistência.

Temos, por isso, de começar por o fazer e por conhecer ainda melhor o meio africano, tanto urbano como rural, mobilizando pessoal para um inquérito à vida das aglomerações nativas periurbanas e à vida tribal, aos costumes e práticas tradicionais, alimentação, bebidas, cerimónias, ritos, recolhendo os elementos psicológicos locais, mantendo o respeito pelos chefes nativos e líderes naturais, levando-os a colaborar nesses inquéritos, obtendo a cooperação de curandeiros e feiticeiros, distinguindo, entre as práticas e as tradições, as úteis, as inofensivas e as nocivas, para só agir sobre estas últimas.

Na maior parte dos países uma parte desse trabalho é feito pelo sociologista, e terá de sê-lo também em Moçambique.

Assim o demonstra plenamente a experiência realizada na área suburbana de Lourenço Marques, pela sociologista Mrs. Hilary Flag, que, em colaboração com alguns enfermeiros auxiliares, que instruiu durante alguns meses, acaba de fazer uma prospecção de tumores, o que representa uma consoladora experiência.

Os principais capítulos em que esta actuação terá de desenvolver-se, cada um deles desdobrando-se em inquéritos e princípios de acção sobre que deverá incidir a acção do educador sanitário, são, pela ordem da importância:

1 — Higiene geral — Luta contra as doenças transmissíveis;
2 — Saneamento — Água potável — Desinsectização — Desinfecção da água;
3 — Estatística vital;
4 — Inquérito socioeconómico — Desenvolvimento agropecuário — Principais medicamentos de alcance social;
5 — Recursos alimentares — Hábitos alimentares — Estudo da nutrição — Doenças carenciais — Alimentação e Economia;

6 — Protecção materno-infantil — Assistência ao parto, assistência pré-natal, primeira infância, mortalidade infantil, kwashiorkor — Puericultura — Vacinas — Soros;
7 — Inquérito às unidades tradicionais de assistência, matrona, curandeiro e possibilidades da sua colaboração ou instrução, aprendizagem técnica;
8 — Situação social dos actuais trabalhadores de assistência, enfermeiros, parteiras e forma de a melhorar.

Apontada esta orientação e entendida por bem necessária a existência do educador sanitário, poderemos para o efeito utilizar o único instrumento flexível que temos à nossa disposição — a Escola Técnica dos Serviços de Saúde, onde funcionam alguns cursos, aos quais se juntaria mais um curso, o de educadores sanitários e o de educadores de Saúde Pública.

Este novo curso teria:

1 — A duração de 2 anos;
2 — Para a sua admissão, deve ser feito um concurso de provas públicas entre os diplomados com o Curso Normal de Enfermagem, com a média mínima de 15 valores; e
3 — De ser fixado como conhecimentos gerais a exigir nas provas de concurso:

Biologia Geral — Matemáticas;
Patologia;
Doenças contagiosas;
Organização social;
Actividades médicas e Assistência e Profilaxia;
Redacção e Exposição Oral de temas sanitários.

Por este concurso se operaria assim uma primeira selecção entre candidatos que já têm uma profissão e que tentam um escalão mais alto.

O curso, além de desenvolver os conhecimentos já mencionados nele, serão professadas as seguintes disciplinas:

Biologia geral — Antropologia — Fisiologia — Genética — Bacteriologia — Patologia — Doenças contagiosas — Profilaxia e Higiene

— Grandes flagelos — Doenças Venéreas — Conhecimentos matemáticos — Estatísticas — Demografia — Psicologia — Sociologia — Organização das sociedades nativas — Biotipologia — Temperamentos mórbidos e predisposições — Ecologia — Móveis fundamentais da conduta humana em geral e da conduta dos rurais — Factores sociais e culturais das sociedades nativas — Campanhas administrativas — Culturas — Gado — Estradas — Silvicultura — Água e irrigação — Mecanização — Comércio — Economia — Alimentação — Nutrição — O frio e o calor — Os pólens — Despiste de doentes — Registos e relatórios — Princípios de higiene e salubridade — Alcoolismo — Intoxicações — Princípios de saúde mental — Medidas de urgência — Desportos — Prevenção contra acidentes — Acidentes de trabalho — Acidentes em casa — Resfriamentos — Questões sexuais — Principais campanhas sanitárias — Paludismo — Tuberculose — B. C. G. — Métodos pedagógicos de educação sanitária — Cartazes — Gráficos — Conferências — Emissões radiofónicas — Organização das associações rurais — Cooperação da população — Cooperação dos organismos administrativos — Técnica da palestra e da publicidade, cartas a particulares, bibliografia, organização de conferências e reuniões.

Durante o curso haverá treinos nos hospitais, nos dispensários, nas delegacias e centros de saúde próximos. Organizar-se-ão grupos de discussão, de que participarão doentes e pessoal são.

Os alunos farão exercícios verbais e escritos sobre temas educativos e demonstrações práticas ao alcance da mentalidade dos rurais e serão treinados na preparação de refeições com os géneros locais e elaboração de tabelas.

Para leccionar e orientar estes cursos dispomos, já preparados, de médicos que professaram cursos de educação sanitária em Londres, um médico que professou um curso de Estatística na América do Norte, um engenheiro que professou um curso de Saneamento em Londres, médicos que professaram cursos de Doenças endemo-epidémicas e de saúde mental, enfermeiros e visitadoras que fizeram cursos de aperfeiçoamento no Brasil e noutros países.

Há que ter em conta que educação sanitária significa participação activa do educando, que deverá, por isso, colaborar tomando parte nos inquéritos, auxiliando nas demonstrações, organizando até associações benévolas para dar impulso a medidas de profilaxia e higiene. E ter mais em conta que as técnicas educativas a adoptar deverão estar em relação com a idade, o sexo, a cultura e a predominância das doenças.

Que o educador sanitário trabalhará em estreita colaboração com o médico, os enfermeiros, o missionário, o administrador, o professor, os régulos, as pessoas influentes, as associações desportivas, os sindicatos, etc.

E que o educador sanitário deverá:

1 — Saber o que é preciso;
2 — Saber o que se pode fazer;
3 — Criar organismos cívicos pró-higiene e trabalhar com os existentes;
4 — Estar à disposição de quem o procure;
5 — Contactar os trabalhadores industriais, os agricultores, os estudantes, as donas de casa.

Os programas dos cursos a professar serão elaborados em cada época, tomando em consideração os problemas mais urgentes e os meios disponíveis. Cada mês terá a sua campanha, sobrepondo-se aos problemas permanentes.

A escola ministrará a técnica das palestras e dará radioemissões.

A escola terá uma relação completa de médicos, parteiras, enfermeiros, dentistas, farmacêuticos, professores, missionários, funcionários administrativos, veterinários, organismos sanitários, pessoal dos laboratórios, grupos de estudos e porá em contacto os alunos com estes futuros pontos de apoio.

A escola organizará um grupo móvel com um médico, um ajudante de radiologia, um educador sanitário, um analista auxiliar. O grupo móvel projectará filmes, terá um aparelho de telefonia, material de propaganda e alto-falante. Fará análises, testes de hemoglobina, radiografias do tórax, análises de urinas, questionários, demonstrações.

O grupo móvel será apetrechado de uma cozinha ambulante para demonstrações dietéticas.

Com base nas noções adquiridas no curso da Escola Técnica dos Serviços de Saúde e nos inquéritos permanentes que o sociologista, o estaticista e os outros professores de curso realizam em zonas-pilotos anualmente escolhidas, o educador sanitário vai actuar sobre vários sectores da população e para cada um deles escolhe um programa de ensino adequado ao seu nível mental e necessidades.

As actividades do educador sanitário obedecem assim a um programa estabelecido, escolhendo-se em cada época um objectivo determinado.

Damos esquemàticamente a nota de algumas dessas actividades:

1 — Visita aos domicílios, pensões, restaurantes, hotéis populares, vendas, talhos. Demonstração de algumas técnicas de preparação de alimentos;
2 — Visita de alojamentos colectivos e inquérito sobre esgotos, fossas, lavabos;
3 — Contacto com os manipuladores de alimentos, padarias, cozinheiros, fábricas de refrigerantes, bares, bazares, para discussão das doenças disseminadas pelos alimentos. Luta contra o alcoolismo e toxicomanias;
4 — Visita de oficinas e fábricas — Palestras sobre atitudes viciosas e acidentes de trabalho. Vestuário — Alojamento — Ginástica — Acidentes da circulação — Fadiga — Resfriamentos;
5 — Visitas às mães — Puericulturas;
6 — Visita à aldeia: Economia culinária, abastecimento de água — Reunião de pessoas influentes — Limpeza de quintais — Retretes — Antipalúdicos — Insecticidas — Luta contra o charlatanismo — Luta contra o cancro — Vacinação — Doenças transmissíveis — Tuberculose — Lepra;
7 — Visita às grávidas — Higiene pré-natal;
8 — Visita às parteiras não diplomadas — Sua colaboração;
9 — Visita às escolas — Ortopedia dos adolescentes — Higiene sexual — Feridas — Queimaduras — Estimulantes;
10 — Associação das escolas, famílias e organismos sanitários — Criação de centros rurais de protecção materno-infantil — Palestras sobre doenças cardíacas.

Este âmbito de acção teòricamente ilimitado é reduzido a um plano prático, variável segundo as condições económicas e sociais das regiões e executado com o auxílio das instituições existentes, entre as quais cumpre salientar as missões católicas.

Mas ao lado do carácter criador do educador sanitário, pela aquisição de noções e atitudes em favor da profilaxia e higiene, ele vai servir para travar, numa sociedade em evolução, alguns malefícios graves da publicidade malsã, da intoxicação por drogas, da tendência ao abandono das terras, abuso de alimentos, abandono de hábitos sadios e de um certo espartanismo da vida. Haverá fàcilmente uma transposição da educação sanitária para os planos social e económico e até ético. O educador sanitário ajudará as campanhas de criação de animais e de peixes, introdução de novas espécies de culturas e de árvores de fruto.

Assim procederá, com vista a auxiliar a resolução do problema alimentar dos nativos, promovendo que se criem proteínas de alto valor biológico, de indiscutível necessidade, pois que sem elas o desenvolvimento físico é precário e o equilíbrio fisiológico não se mantém de forma desejável.

E assim, ao lado da acção educativa sobre todos os aspectos, fará fàcilmente propaganda dos preceitos de higiene, pois que o nativo é ingènuamente confiante e aceitará bem as noções de higiene e, ao lado destas, noções de profilaxia das doenças evitáveis.

Devemos desde já frisar, porém, que estas campanhas não são o verdadeiro objectivo do educador sanitário, que é circunscrito. O educador sanitário é um auxiliar das campanhas sanitárias e tem de limitar-se a isso. Só acidentalmente ajudará nas outras campanhas, quando elas tenham uma finalidade sanitária. A sua função é tratar do problema cuja solução está confiada aos Serviços de Saúde, embora em colaboração com outros Serviços.

E por isso, em cada ano, os Serviços de Saúde organizarão uma lista dos temas que cumpre desenvolver para a protecção da saúde pública.

É intuitivo que o educador sanitário será ao mesmo tempo um investigador. Sendo diferentes os níveis de cultura sanitária, quer dos indivíduos que compõem um aglomerado quer das diferentes regiões, haverá uma necessidade comum em cada grupo, em cada zona e um método prático de a satisfazer. Esta investigação compete ao educador, que proporá aos Serviços a técnica da sua realização. Haverá, assim, ou não, depois da campanha educativa, uma mudança sensível no comportamento da população. A não haver, haverá que utilizar método diferente. Empregar-se-á o questionário, a observação familiar, a ficha sanitária preenchida pelo educador, o interrogatório posterior à palestra e à exposição gráfica.

Será talvez ainda prematuro pensar-se nos meios de acção em grande escala.

Uma tentativa, porém, de muito alcance será pôr ao lado da maternidade uma creche e interessar numa e noutra a comunidade, saindo do sistema actual em que a maternidade é *só* um *Organismo* do *Estado*.

A creche e a maternidade são, em meu entender, os vigamentos da educação sanitária e ajudarão as mães a solucionar problemas de saúde e de economia.

A comprová-lo temos, em plena actividade e fora já do regime experimental, 4 unidades na Província, onde o processo tem plena utilização

A creche constituirá uma unidade com a família, onde se prolongarão os ensinamentos ministrados nela. As mães adquirirão nas creches noções sobre a alimentação, saúde e aleitamento, ouvirão palestras, assistirão a exposições e filmes.

O exame oncológico precoce ajudará a luta contra o cancro e o radiorrastreio dará noções antituberculosas.

A prospecção a tempo e horas dará elementos indispensáveis na defesa da saúde contra a lepra e a doença-do-sono.

A prospecção e uma vigilância aturada das populações no que respeita à ancilostomíase, bilharziose, boubas, úlceras fagedénicas, micoses, doenças venéreas, darão de igual modo elementos seguros na luta contra estes verdadeiros flagelos da raça negra.

À campanha antiboubática maciça, presentemente em plena execução no distrito da Zambézia, vamos nós buscar, de resto, além de outras vantagens, a utilidade indiscutível da excelente oportunidade de treino de pessoal auxiliar para outras actividades médicas e esperamos mesmo ainda, nos anos subsequentes, ao longo da execução da campanha em curso, utilizá-la simultâneamente para combate a outras doenças.

Só assim, de resto, a defesa da saúde terá sucesso e o bem-estar dos indivíduos aparecerá como condição indispensável do bem-estar colectivo.

A Escola será o centro de documentação científica necessária como base da educação e nela se apoiarão os educadores sanitários.

A Escola publicará livros e brochuras de educação sanitária e um vocabulário de termos médicos para uso dos educados.

Na Escola professar-se-ão assim cursos e lições práticas, com a única finalidade de pôr em actividade este notável método da educação sanitária, que, com base na formação dos educadores sanitários, conta com a formação do educando, essencial participante do método, criador da melhoria das condições de vida do nativo, amparando-o, guiando-o, em tudo o que represente desenvolvimento da sua economia e criação de riqueza.

Damos, em anexo, alguns programas de cursos e lições práticas, destinados à finalidade que nos propomos.

ANEXO

PROGRAMA DE CURSOS E LIÇÕES PRÁTICAS

I — CURSOS PRÁTICOS PARA ADOLESCENTES DO SEXO FEMININO — CRIADAS — VIGILANTES DE CRIANÇAS

Idade mínima — 15 anos.
Habilitações — ler e escrever.
Duração — 20 dias.
Teoria — mínima.
Prática — 2 horas por dia.

1 — Cuidados práticos às crianças:

O quarto da criança
O enxoval.
Desenvolvimento — Hábitos — Brinquedos — Exercícios — Sinais de doença — Profilaxia dos acidentes.
Banho.

2 — Alimentação:

- Preparação.
- Perturbações gastrintestinais.
- Desmame.
- Dietas fundamentais.
- Dentição.

3 — Enfermagem simples:

- Cama.
- Levantar do doente.
- Banho de esponja.
- Saco de gelo e água quente.
- Clisteres.
- Alimentação na cama.
- Dejecções na cama.

II — CURSOS PARA CASAIS

1 — Noções fundamentais das condições físicas, mentais, emocionais e económicas do casal.
2 — Genética — Eugénica.
3 — Nutrição.
4 — Higiene geral na casa na aldeia.
5 — Orçamento.
6 — Sinais de gravidez.
7 — Evolução da criança.
8 — Sinais de perigo.
9 — Cuidados pré-natais.
10 — Dietário — Cálcio — Proteínas — Vitaminas — Sais minerais.
11 — Exercícios físicos.
12 — Respiração abdominal e torácica.
13 — Preparativos do parto.
14 — Visitas dos hospitais e maternidades.
15 — Parto domiciliário.
16 — Mobiliário para a criança.

17 — Roupas e sua mudança.
18 — Banho.
19 — Biberões — Fórmulas e alimentares.
20 — Responsabilidades legais — Registo Civil.
21 — Vacinas.

III — CURSO PRÉ-NATAL

A) Anatomia e Fisiologia da reprodução:

1 — Sinais de gravidez.
2 — Fecundação.
3 — Evolução do feto.
4 — Data do parto.

B) Higiene da gravidez:

1 — Higiene sexual.
2 — Vestuário.
3 — Ginástica.
4 — Banho.
5 — Regras.
6 — Cuidados dentários.
7 — Relações conjugais.
8 — Higiene dos seios.
9 — Tabaco — Álcool — Superalimentação.
10 — Sono — Repouso.

C) Assistência médica à grávida:

1 — Pesagem.
2 — Tensão arterial e pulso.
3 — Exame de sangue — Factor *RH*-Teste de hemoglobina e hemograma.
4 — Exame do ventre.
5 — Urinas.
6 — Toque e exame ginecológico.
7 — Exame da pelve.
8 — Exame geral.
9 — Incómodos da gravidez.

10 — Incómodos a relatar ao médico.
11 — Hemorragias.
12 — Pruridos — ardores.
13 — Obnubilação visual.
14 — Dores abdominais.
15 — Vómitos menores e incoercíveis.
16 — Calafrios.
17 — Febre.
18 — Cefaleias.
19 — Ruptura súbita da bolsa.

D) Regime alimentar:

1 — Alimentos básicos.
2 — Amostras alimentares.
3 — Variações do peso materno.
4 — Ementas e suas variações no decurso da gravidez.

E) Trabalho de parto:

1 — Preparativos do parto.
2 — Sinais precursores. Contracções uterinas, seu ritmo e anomalias.
3 — Ruptura da bolsa.
4 — Descarga mucosa.
5 — Parto.

    a) Dilatação do colo.
    b) Expulsão da criança.
    c) Expulsão da placenta.

7 — Filmes.

F) Assistência à puérpera:

1 — Puerpério.
2 — Involução.
3 — Seios e suas alterações.
4 — Repouso e exercício.

5 — Dieta.
6 — Eliminação.
7 — Composição do leite.
8 — Lactação e galactogenos.
9 — Restabelecimento completo da mãe.
10 — As primeiras 6 semanas.
11 — Relações sexuais após o parto.
12 — Crendices e superstições.
13 — Menstruação após o parto.
14 — Aleitamento e concepção.
15 — Aleitamento e menstruação.
16 — Intervalo normal entre a gravidez e suas relações com a saúde da mãe, da criança, do casal e com economia.

G) O neonato:

1 — Visita à maternidade — Necessidades fundamentais do neonato.
2 — Vestuário.
3 — Equipamento de uma maternidade, dum infantário e duma creche.
4 — Cuidados físicos com a criança.
5 — Sinais de saúde.
6 — Sinais de sono.
7 — Reflexos e atitudes.
8 — Choro.
9 — Indicações de chamada de médico.
10 — Banho.

H) Crítica — Discussão — Sugestões — Informações de particularismos locais.

IV — CURSO DE FISCAIS DE GENEROS ALIMENTARES

1.ª lição

1 — Introdução — Noções gerais de doenças transmissíveis.
2 — Bacteriologia.

3 — Meios de cultura — Água — Solo — Alimentos.
4 — Cozinha, retretes, detritos, alimentos em decomposição, moscas.
5 — Micróbios úteis.
6 — Micróbios nocivos.
7 — Micróbios inofensivos.
8 — Portas de entrada da infecção.

2.ª lição

1 — Habitação.
2 — Ventilação e luz.
3 — Casa de jantar, rede mosquiteira, lavatórios, frigorífico.
4 — Cozinha, fogão, receptáculos de lixo, esterilização de água.
5 — Retretes.
6 — Protecção de alimentos.
7 — Armazenagem de géneros: peixe, carne, ovos, legumes, hortaliças.
8 — Mercearias, cozinhas, cozinheiros, ajudantes, mesas de cozinha e utensílios.
9 — Criados, cadeiras, toalhas, pratos, copos.

3.ª lição

Portadores de germes e defesa:

1 — Moscas — Protecção mecânica de alimentos, hortaliças, luta contra a humidade e poluição, insecticidas.
2 — Baratas — Destruição pela água quente — *Habitat.*
3 — Ratos — Protecção mecânica, esconderijos, ratoeiras, fome.
4 — Cooperação das pessoas com as autoridades sanitárias.

4.ª lição

1 — Legislação sanitária.
2 — Infracções frequentes, contravenções internacionais — Delitos graves.
3 — Certificado de saúde.
4 — Protecção de alimentos.
5 — Alimentos impróprios para consumo — Falsificações.
6 — Exposição de alimentos e cuidados necessários. Lixo e detritos — Resíduos da cozinha e do restaurante.
7 — O papel da água quente.
8 — Higiene das despensas e armazéns.
9 — Vizinhanças incómodas — Fumos — Cheiros.
10 — Papel de embrulho.

V — CURSO PRÁTICO DE AUXILIARES SANITÁRIOS DAS DELEGAÇÕES DE SAÚDE

Orientação

1 — Definição e exemplos de saúde pública.
2 — Missão dos delegados de Saúde.
3 — Orgânica das delegações de Saúde.
4 — Papel do auxiliar.
5 — Equipa sanitária.
6 — Leis e regulamentos.
7 — Ética profissional.

Objectivos

1 — Fiscalização dos esgotos.
2 — Higiene da água.
3 — Esterilização do leite.
4 — Higiene das retretes, lavabos, mictórios, escarradeiras, fossas, nos estabelecimentos públicos, lixeiras, estrumeiras, nitreiras, quintais.

5 — Restaurantes.
6 — Hotéis e pensões.
7 — Teatros e cinemas.
8 — Mercados.
9 — Escolas.
10 — Barbearias, lavandarias.
11 — Padarias, casas de chá, bares.
12 — Habitações.
13 — Luta contra os animais nocivos, insectos e ratos.
14 — Higiene industrial, fumos, cheiros pestilenciais.
15 — Vizinhança incómoda.

Investigação

1 — Métodos gerais.
2 — Métodos especiais.
3 — Colheita de amostras, alimentos, água.
4 — Relatórios.

Inspecção

Restaurantes:

1 — Pessoal.
2 — Cozinha.
3 — Casa de jantar.
4 — Lavabos e retretes.

Padarias:

1 — Instalações.
2 — Armazém e despensa.
3 — Balcão e loja.
4 — Lavabos.

Doçarias — Confeitarias — Leitarias:

1 — Pessoal.
2 — Fabrico.
3 — Loja.
4 — Lavabos.

Refrigerantes — Sorvetarias:

1 — Pessoal.
2 — Fabrico de xaropes.
3 — Engarrafamento.
4 — Lavagens
5 — Armazéns.
6 — Lavabos.

Mercados:

1 — Higiene de vendedores.
2 — Detritos e lixos.
3 — Tábuas e tabuleiros.
4 — Protecção contra as moscas.

Barbearias — Institutos de Beleza:

1 — Pessoal.
2 — Sanidade.
3 — Salões.
4 — Tráficos ilícitos.

Teatros e Cinemas:

1 — Pessoal.
2 — Salas.
3 — Parasitas e Insectos.
4 — Ventilação.
5 — Lavabos.
6 — Bastidores.
7 — Iluminação.
8 — Protecção contra incêndios.

Hotéis e pensões:

1 — Pessoal.
2 — Quartos.
3 — Lavabos.
4 — Casas de banho.

Piscinas públicas:

1 — Cloro residual.
2 — Protecção contra acidentes.
3 — Chuveiros — vestiários.
4 — Lavabos.

Drogarias:

1 — Loja.
2 — Esterilização e lavagem de garrafas.
3 — Lavabos.
4 — Livros e escrituração.
5 — Utensilhagem.
6 — Sanidade.

Residências particulares:

1 — Água.
2 — Lavabos, casas de banho, retretes.
3 — Fossas.
4 — Quartos e cubagem.
5 — Pavimentos e tectos.
6 — Lixo e detritos.
7 — Sanidade.

Luta contra animais:

1 — Ratos.
2 — Moscas.
3 — Mosquitos.
4 — Percevejos
5 — Pulgas.
6 — Baratas.

PRATICA DE INSPECÇÃO

1 — Lição sobre a observação e ensino dos manipuladores de alimentos. Razões dos regulamentos sanitários. Oportunidade e técnica de contacto entre os auxiliares oficiais e os empregados das casas particulares.

2 — Lição sobre filmes, projecções, cartazes, panfletos, gráficos, boletins.
3 — Lição sobre organização de reuniões públicas, associações pró-higiene, competições, prémios.
4 — Lição sobre campanhas de imunidade. Construção de retretes e fossas. Limpeza periódica.
5 — Lição sobre progresso profissional, biblioteca, conferências, investigação, intercâmbio — Discussão — Sugestões críticas.

LIÇÕES DE PUERICULTURA

A) Introdução:

1 — Objectivos.
2 — Vantagens.
3 — Equipa dos Serviços de Saúde.
4 — Doenças gastrintestinais.
5 — O que a mãe pode fazer.

B) Bacteriologia:

1 — Patogénicos.
2 — Não patogénicos e úteis.

C) Higiene pessoal:

1 — Geral.
2 — Cabelo.
3 — Olhos.
4 — Ouvidos.
5 — Dentes.
6 — Face.
7 — Vestuário.
8 — Repouso e sono.
9 — Exercício e recreio.
10 — Filmes.

D) Nutrição:

1 — Mulher na vida ordinária activa.
2 — Grávidas.
3 — Período de lactação.
4 — Cuidado com os seios.
5 — Vantagens e inconvenientes do biberão.
6 — Filmes.

E) Alimentação e cuidados ao infante:

1 — Higiene infantil.
2 — Crescimento.
3 — Imunização B.C.G.
4 — Alimentação ao peito.
5 — Alimentação artificial.
6 — Mista.
7 — Complementar.

F) Doenças da 1.ª infância:

1 — Gastrintestinais.
2 — Pele.
3 — Respiratórias.
4 — Nutricionais.
5 — Parasitoses.
6 — Febres eruptivas.

G) Sanidade ambiente:

1 — Casa.
2 — Aldeia.
3 — Filmes.

Lições sobre economia doméstica e alimentar:

1 — Introdução.
2 — Revista geral dos hábitos alimentares — Importância da nutrição — Filmes.

3 — Alimentos básicos.
4 — Plano de uma refeição:

  a) Ementa;
  b) Vitaminas e sais, sua conservação;
  c) Mercados e preços.

5 — Prática culinária de uma refeição.
6 — Alimentação e nutrição da mãe:

  a) Relação de saúde entre mãe e filho lactente;
  b) Erros e preconceitos alimentares;
  c) Necessidades alimentares das mulheres normais;
  d) Das grávidas;
  e) Dietário durante a gravidez e lactação;
  f) Dietário durante as doenças da gravidez;

7 — Nutrição infantil:

  a) Valor da amamentação;
  b) O essencial na alimentação infantil;
  c) Alimentação artificial da criança;
  d) Alimentação complementar;
  e) Demonstrações e exercícios.

8 — Nutrição dos pré-escolares e escolares.
9 — Plantas e animais da região e seu valor nutritivo:

  a) Teoria;
  b) Projecções.

10 — Preparação de alimentos:

  a) Teoria;
  b) Prática.

11 — Preparação de dietas normais, de gravidez e de lactação.
12 — Orçamento.
13 — Plano de uma cozinha económica:

- *a*) Quesitos escritos;
- *b*) Prática;
- *c*) Visitas domiciliárias.

VI — CURSO DE EDUCAÇÃO SANITÁRIA PARA ENFERMEIRO PROFISSIONAL

1 — Introdução.
2 — Orgânica dos Serviços de Saúde e objectivos.
3 — Educação sanitária:

- *a*) Definição;
- *b*) Conceito;
- *c*) Objectivo;
- *d*) Princípios;
- *e*) Método.

4 — História de educação sanitária:

- *a*) Na antiguidade;
- *b*) Em Portugal;
- *c*) Em Moçambique.

5 — Instrumentos de educação sanitária:

- *a*) Imprensa;
- *b*) Rádio;
- *c*) Televisão.

6 — Principios de educação:

- *a*) Respeitar interesses;
- *b*) Focar a atenção;
- *c*) Associar causas e efeitos.

7 — Educação sanitária do público:

- *a*) Contactos pessoais;
- *b*) Técnicos de propaganda.

8 — Materiais de ensino:

- *a*) Locais acessíveis;
- *b*) Demonstrações.

VII — LIÇÕES PARA CASAIS

1 — Reuniões de aldeia e organização do curso.
2 — Higiene e sanidade de uma aldeia.
3 — Combate de doenças contagiosas.
4 — Menstruação.
5 — Gravidez.
6 — Cuidados pessoais com a gravidez.
7 — Nutrição normal.
8 — Nutrição da gravidez e lactente.
9 — Cuidados dentários da grávida.
10 — Preparativos para o parto.
11 — Sinais de parto.
12 — Cuidados pós-natais à mãe.
13 — Cuidados com o neonato e sua alimentação.
14 — Banho e vestuário.
15 — Biberão e fórmulas alimentares.
16 — Crescimento da criança.
17 — Higiene social.
18 — Doenças habituais da primeira infância.
19 — Assistência social — Filmes.

VIII — LIÇÕES PARA ADOLESCENTES

1 — Introdução.
2 — Desenvolvimento físico na adolescência.
3 — Menstruação.
4 — Factores funcionais da adolescência.
5 — Nutrição normal e factores do crescimento.
6 — Relações sociais — Personalidade.
7 — Higiene pessoal.
8 — Higiene comunal.
9 — Doenças contagiosas.
10 — Cuidados dentários.
11 — Enfermagem doméstica — primeiros socorros.
12 — Filmes.
13 — Trabalhos práticos.

IX — LIÇÕES PARA NOIVAS

1 — Sinais de gravidez.
2 — Higiene de gravidez.
3 — Preparativos do parto.
4 — Visitas a maternidades.
5 — Serviços sociais.
6 — Temperatura, termómetro.
7 — Banho da criança.
8 — Clisteres à criança.
9 — Menstruação e lactação.
10 — Fisiologia e crescimento do feto.
11 — Alimentação artificial.
12 — Alimentos básicos.
13 — Nutrição materna.
14 — Nutrição fetal.
15 — Doenças da primeira infância.
16 — Doenças contagiosas.
17 — Higiene dentária.
18 — Orçamento.

19 — Planos culinários.
20 — Nutrição na idade pré-escolar.
21 — Trabalhos manuais.

X — LIÇÕES PARA MANIPULADORES DE ALIMENTOS

1 — Principais alimentos — sem empobrecimento ou enriquecimento.
2 — Filmes.
3 — Doenças de origem alimentar.
4 — Alimentos — meios de cultura.
5 — Protecção de alimentos.
6 — Plano de uma refeição.
7 — Princípios de alimentação colectiva.
8 — Filmes.
9 — Arroz — mandioca — milho — feijão — mapira.
10 — Carne — Peixe — Amendoim — Leite — Coco — Mafurra.
11 — Técnicas de preparação de alimentos.
12 — Amostras de ementas.
13 — Economia alimentar.
14 — Saladas — Frutas — Citrinas.
15 — Alimentos e saúde pública.

XI — PROGRAMA DE ENSINO SANITÁRIO ESCALONADO EM 6 ANOS, PODENDO FUNCIONAR NAS ESCOLAS

1.º e 2.º anos

1 — Higiene do corpo:

Mãos;
Rosto;
Dentes;
Nariz;
Ouvidos;
Cabelo;
Pés.

2 — Higiene do vestuário.
3 — Limpeza da escola e cercanias:

Refeitório;
Lavabo;
Pátio;
Lixo e detritos.

4 — Atitudes viciosas:

De pé;
Na marcha;
Na posição sentado.

5 — Sabão — Escovas.
6 — Comer à mesa:

Colher;
Garfo e faca;
Toalha;
Pratos.

Estas noções são produzidas de forma prática, com inspecção e fiscalização diária da higiene pessoal dos alunos. É útil estabelecer medalhas, prémios, menções honrosas. Vigilância constante dos lavabos, do pátio pelo professor, informando os alunos do seu uso e criando hábitos de asseio e higiene.

3.º ano

1 — Hábitos atrasados e seus perigos:

Pele;
Olhos;
Ouvidos;
Dentes;
Cabelos.

Sublinhar as doenças e os incómodos provados pela falta de higiene pessoal. Pele e suas infecções comuns. Limpeza de feridas e seus cuidados.

Olhos e ouvidos. Importância da sua higiene e perigos de infecção pelas roupas sujas e mãos. Óculos. Corpos estranhos.

2 — Vestuário. Distinção entre conforto e luxo.
3 — Posições normais e anormais — respiração correcta — Fadiga.
4 — Posições na leitura e na escrita.

Acentuar a influência das posições viciosas sobre o esqueleto, órgãos digestivos, respiração, boas e más.

5 — Hábitos-Alimentos:

Regularidade das refeições.
Importância do pequeno almoço, evitar comprar alimentos nas ruas.
Géneros alimentares.
Função dos alimentos.
Leite natural — condensado — seco — nata.
Frutas frescas.
Legumes verdes.
Doçaria e seus inconvenientes.
Água potável.
Doenças hídricas.
Mastigação.

6 — Exercício — Repouso — Fadiga:

Jogos e seu valor.
Ar livre — educação física.
Fadiga, suas origens — Falta de alimentos.
Falta de sono.
Necessidade de dormir cedo.

7 — Limpeza da escola e cercanias:

Ar puro — Sol — Sua importância no crescimento — Combate aos ratos, baratas, moscas e seu papel na defesa da saúde.
Interessar a criança na higiene da escola e pátios.

8 — Como evitar as doenças mencionadas no programa. Revisão da matéria dos 3 anos.

4.º ano

Alimentação

1 — Géneros e tipos de alimentos — Seu papel no organismo:

Protectores contra as doenças.
Produtores de energia.
Estimulantes do apetite.
Água.
Discussão sobre a alimentação habitual de cada criança, evitando os termos técnicos.
Evitar os termos técnicos.
Concentrar a atenção sobre os alimentos acessíveis.

2 — Classificação dos alimentos:

Necessidades alimentares quotidianos.
Alimentação variada e sua importância na robustez, compleição, atletismo, desporto.

3 — Alimentação equilibrada — Fruta — Legumes.
4 — Leite em pó e seu valor — Preparação do leite e sua medição — Leite condensado.

5 — Origem dos alimentos — Horta — Searas:

Gado — Lacticínios — Farinação.
Alimentação dos animais.

Mamíferos e o papel do leite na sua criação.
Visitas aos mercados e hortas.
Técnicas da protecção da carne, dos legumes, dos peixes, das farinhas.
Protecção mecânica — Frigoríficos.
Leite — Alimento completo para a 1.ª infância e apropriado para todas as idades.

6 — Água:

Água potável, sua captação e protecção.
Inquinação — Poços inquinados.
Tanques, ribeiras, charcos.
Visitas aos locais de captação — condutas.
Torneiras.
Filtros — Fervura.

7 — Alimentos e sua limpeza:

Sítios próprios para guardar alimentos.
Lavagem das mãos — Visitas aos refeitórios.
Louça — utensílios de cozinha.
Lavagem de roupa.

8 — Digestão — Mastigação — Protector da digestão:

Vantagem de comer lentamente.

9 — Evacuação — Obstipação:

Latrinas — sua higiene.

10 — Revisão.

5.º ano

Doenças causadas por falta de higiene e asseio.

1 — Dermatoses:

Sarna;
Tinha;
Feridas;
Úlceras;
Como reconhecer estas doenças;
Processos de disseminação destas doenças;
Como as tratar;
Explicação sobre germes, doenças infecciosas, sua transmissão directa e pelo vestuário.

2 — Doenças espalhadas pela tosse e escarros:

Tuberculose;
Gripe;
Pneumonia;
Varíola;
Sarampo;
Coqueluche.

Explicar como estas doenças se propagam e como se evitam. Sublinhar que tossir sem levar a mão à boca e escarrar são hábitos perigosos para a saúde dos outros. Explicar que estas doenças se transmitem por:

Promiscuidade;
Escarrando no chão;
Poeiras e detritos portadores de germes.

3 — Doenças transmitidas pelas fezes:

Anquilostomíase;
Febre tifóide;
Disenteria;
Como se transmitem estas doenças;
Como as tratar;
Inquinação das unhas pelas fezes. Os manipuladores de alimentos com as unhas poluídas trans-

mitem as doenças de origem intestinal. Padeiros. Cozinheiros;
Transmissão pelas fezes que inquinam as águas — Ribeiros — Poços;
Água potável — Fervura;
Excremento que serve de adubo aos legumes-transmissão pelas saladas. Lavagem de legumes e saladas;
Moscas e ratos que se alimentam de fezes;

Prevenção destas doenças por:

Limpeza individual;
Utilização correcta das retretes;
Protecção de água potável;
Limpeza das mãos — Manipulação de alimentos;
Protecção contra moscas, ratos, poeiras;
Cozedura de legumes;
Combate à mosca;
Ciclo de transmissão de anquilostoma — Sua entrada no corpo pela pele e localização no duodeno;
Importância do calçado — Higiene das latrinas para evitar o anquilostoma.

4 — Doenças transmitidas pelos insectos:

Piolhos;
Percevejos;
Pulgas;
Mosquitos;
Moscas;
Baratas;
Seu tratamento — Profilaxia;
Revista de lição de higiene corporal;
Higiene do cabelo — Vestuário — o leite;
Dificuldades de reprodução dos piolhos e percevejos nos meios asseados;

Dermatoses provocadas pelos piolhos e pelas unhas sujas;
Febres, insónias, pruridos provocados pelos percevejos;
Origem de paludismo, medidas públicas antimaláricas. Mosquiteiro.

5 — Doenças transmitidas pelos animais doentes e sua propagação:

Ténia;
Tuberculose;
Intoxicações alimentares.

6 — Água potável:

Fontanários, poços, ribeiras, chuvas;
Discussão sobre a água da Escola, meios para a sua esterilização.

7 — Evacuação dos detritos — Doenças originadas por eles.
8 — Limpeza da Escola e cercanias — Refeitório — Ar fresco e poluído:

Observações;
Moscas portadoras de germes;
Moscas e excrementos infectados, feridas;
Alimentos e bebidas;
Leite de animais doentes — Leite de vaca e cabra e sua inquinação;
Caixas de lixo e sua importância;
Exame e discussão do asseio da Escola e das alunas;
Aeração — Limpeza dos pavimentos, móveis, paredes, utensílios de cozinha, refeitório, alimentos, pessoal de cozinha, limpeza do guarda-comidas.

9 — Protecção de alimentos — Revisão e discussão:

Comidas dos restaurantes sujos e vendedores ambulantes.

10 — Regras elementares para a boa saúde:

Ar fresco, higiene, alimentação, exercício, repouso.

11 — Revisão da matéria dos anos anteriores.

6.º ano

Nutrição:

1 — Revista dos principais assuntos relacionados com a alimentação.
Empregar alguns termos técnicos, como proteínas, vitaminas.
Classificar os alimentos locais. Necessidades alimentares. Higiene equilibrada. Regime misto. Proteínas. Hidratos de carbono, vitaminas, sais dos alimentos locais. Tabelas.
Valor alimentar do leite em pó desnatado.
Quantidades. Comparação dos vários regimes.
Demonstrações sobre preparação de leite.
Dieta da criança, dos pais, dos irmãos.
2 — Digestão. Aparelho digestivo, suco gástrico e suas funções.
Trajecto dos alimentos. Diafragma e imagens. Quadros murais e flanelgrafo.
3 — Sangue — Sua função — Manter a temperatura fixa — Circulação.
Transportar alimentos. Eliminação dos produtos tóxicos e resíduos — Luta contra os micróbios — Composição do sangue — Oxigénio — Limpeza e poluição do ar.
Funções do coração, artérias, veias, gráficos, desenhos.
4 — Regras gerais de saúde e sua revisão:
Higiene corporal;

Ambiente e sua higiene;
Higiene geral;
Ar fresco, ar frio, sol, exercício;
Repouso — Sono;
Atitudes e posição e seus vícios.

5 — Doenças infecciosas e sua transmissão — Medidas preventivas — Imunização — Vacinação.

6 — Acidentes e primeiros socorros:
Feridas e escoriações;
Queimaduras;
Picadas e mordeduras de insectos;
Corpos estranhos aos olhos, ouvidos, nariz, garganta, estômago.

7 — Farmácia da infância:
Lição especial para raparigas;
Cuidado com os infantes;
Sua alimentação;
Banho;
Roupas — Vestuário da mãe e criança.

8 — Serviço de saúde local:
Actividade oficial na luta contra as doenças infecciosas;
Instalação de água, esgotos, saneamento — Drenagem — Aterros;
Clínicas de puericultura e pediatria — Actividades do médico, dentista, enfermeiro, fiscais, assistentes sociais, parteiras.

Cursos similares serão organizados para os Organismos Desportivos, associações religiosas e forças armadas de acordo com as directrizes fornecidas pelos seus dirigentes.

PROGRAMA DA CADEIRA DE SOCIOLOGIA

1 — Como é constituída a família indígena.
2 — Como é constituído o grupo familiar.
3 — Qual o limite dos poderes do chefe — e qual a liberdade de agir dos membros.

4 — Quais são as relações entre os grupos.
Qual a organização política — pré-nacional — Rei — Corte — dignatários — chefes — alianças — parentescos.

5 — Quais são as relações e como se estabelecem com as autoridades nacionais — Qual a função da organização pré-nacional nesta relação.

6 — Quais são os conceitos religiosos e as práticas religiosas anteriores ao cristianismo.

7 — Quais as práticas e ritos — Noções do universo e do homem.

8 — Ritos religiosos e sociais relativos às doenças — nascimento — morte — casamento — grupos de alianças e não alianças — Funerais.

9 — Meios de subsistência — Rendimento — Repartição do solo.

10 — Despesas ordinárias — e extraordinárias.

11 — Alimentação ordinária e extraordinária — Refeições — Animais domésticos — Possibilidades de criação — Componentes vegetais e animais de alimentação.

12 — Vestuário ordinário e de cerimónia — Seu custo.

13 — Alojamento — Utensílios — Móveis.

14 — Meios de transporte — Ordinários e extraordinários.

15 — Grau de instrução dos componentes — Jornais — Rádio.

16 — Fenómenos jurídicos — Administração da justiça pré-nacional.

17 — Feitiçaria — Magia — Ritos — Símbolos — Intermediários — Consciência colectiva — Tabos — Receios — Medo — Ansiedade.

18 — Instituições pré-nacionais — Concepções médicas.

19 — Medicamentos administrados — de origem tribal — Obstáculos sociais às terapêuticas científicas — Avaliação e elementos a considerar — Fraudes — Abstenção — Reacções pessoais e tribais às assistências na doença e resultados dos métodos nativos — Prevenção — Medicamentos gratuitos e pagos.

20 — Desmame — Crenças e ritos — Alimentos novos — culinária.

21 — Puberdade — Crenças — ritos — Cerimónias — Iniciação.

22 — Delinquência juvenil.

23 — Almas dos mortos — Cerimónias expiatórias — Vinganças dos mortos.

24 — Sangue — urinas e fezes — Importância psíquica das colheitas — Filtros e bebidas mágicas.

25 — Preferem os nativos as habitações de tipo europeu? Porquê? ou porque não?

26 — Noção de distância — Noção do tempo — Noção de simultaneidade — Noção de medida das funções fisiológicas — Noção de geometria.

27 — Autoridade — Nativa — Nacional — Inter-relações.

28 — Presença de europeus na aldeia — Suas relações e reacções locais acerca de agricultores, comerciantes, missionários, professores, médicos — enfermeiros — agrimensores — engenheiros.

29 — Interdições — Alimentares — Lugares — Sexuais — Sociais — de comportamento.

30 — Concepção das doenças:

Origem — Potências sobrenaturais — Quebra de tabos.
Métodos usados para combates.
Afecções em que se consulta o médico, ou o curandeiro.
Feiticeiros — Suas categorias — Existência ou não de escolas de feiticeiros ou curandeiros.
Lista de remédios — Colheita de exemplares — Vocabulário;
Mitologia — Mitos relativos à concepção, parto, doenças, morte, casamento — protecção e profilaxia — comparação com os mitos das outras regiões;
Motivos religiosos de certos alimentos;
Apontamentos sobre as colheitas e gado;
Plantas alimentares;
Carne consumida;
Razões do abandono de certos alimentos;
Processos de conservação dos alimentos — Salmoira — Vinagre — Secagem — Fermentação;
Mercados — Trocas — Internas e externas;
Dote;
Alimentação em relação às estações;

Possibilidades e dificuldades religiosas e sociais nas alterações e introduzir na:
Criação;
Culturas — Barragens de água;
Plantas alimentares — canalização de água;
Retretes;
Evacuação dos dejectos;
Poluição das águas;
Mitologia ligada a estes elementos;
Tratamento das crianças;
Concepção da vida intra-uterina, nascimento, 1.ª infância, mortalidade infantil.

MÉTODOS DE ENSINO PARA EDUCADORES SANITÁRIOS

MARIA AMÁLIA BORGES PITTA [1]

Para ensinar, é necessário um certo número de requisitos, que, sem eles, o nosso trabalho não terá a eficácia desejada.

Tratarei, em resumo, de dois deles:

I) Edifício escolar.
II) Métodos de ensino.

EDIFÍCIO ESCOLAR

Devidamente apetrechado, que constaria de:

a) Gabinete escolar.
b) Gabinete da administração (secretaria).
c) Sala de recepção.
d) Sala-de-estar.
e) Biblioteca.
f) Salas de aulas (em número suficiente).
g) Museu.
h) Sala de gráficos.
i) Gabinete fotográfico.
j) Sala de jantar.
l) Quartos individuais, de preferência.
m) Sanitários.
n) Sala de dietética.
o) Quarto de costura.
p) Lavandaria e quarto de engomados.
q) Quarto de arrumações.
r) Terraço.

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[1] The author did not attend the meeting.

MÉTODOS DE ENSINO

Em Moçambique, quando for introduzido mais este curso, temos primeiramente de ter em vista como e até que ponto o nativo será capaz de ministrar estas aulas, pois será um curso dado mais eficientemente se já for elaborado atendendo à sua mentalidade.

Da minha prática de ensino, tenho verificado que os nativos desta província têm uma memória privilegiada, donde lhes advém a facilidade de decorar com rapidez, chegando até ao ponto de transcreverem textualmente tudo quanto o professor lhes ensinou. Têm, contudo, muita dificuldade de vocabulário e na maioria manifestam uma aversão acentuada à matemática. São algo acanhados e com escassa iniciativa, atrapalhando-se com muita facilidade, e a tal ponto que ficam completamente paralisados. Quando é preciso aplicar a teoria à prática, como geralmente decoram sem perceber, ficam mesmo perturbados.

Tenho notado também que as raparigas são todos os anos piores alunas do que os rapazes, em todo o campo, mesmo no manual.

a) Despertar o interesse da profissão:

1.º — Vamos despertar o interesse do aluno, mostrando o objectivo, que deve ser claramente definido.

b) *O período de adaptação é indispensável,* para que o aluno possa avaliar se tem realmente vocação e o professor verificar se ele será ou não capaz de ser um bom profissional. A selecção será feita dentro dum período de seis meses, pelo menos, por técnicos competentes e conscienciosos, tendo em vista a *qualidade, e não a quantidade,* para que se não tornem elementos prejudiciais à sociedade.

c) *Se existir um número excessivo de horas de trabalho,* diminuirá a capacidade física e intelectual, o aluno não dará o resultado pretendido e, como consequência, desempenhará mal as suas atribuições.

d) *Será um profissional com melhor cultura geral* se possuir o curso geral de enfermagem, visto haver cadeiras relacionadas com os dois cursos, como, por exemplo: higiene, profilaxia e dietética. Poderá mesmo aplicar alguns conhecimentos já adquiridos para melhor desempenhar a sua nova profissão.

PROGRAMA

1.º — *Objectivo:*

a) O objectivo da educação sanitária será:

I) Condições de uma vida sã na escola.
II) Serviços de higiene escolar e serviços sanitários da colectividade.
III) O ensino sanitário.

Estes diferentes aspectos estão estritamente ligados e devem todos os três ser tomados em consideração logo que se examine o género de formação que convém dar aos membros de ensino, para lhes permitir de se ocuparem em devida conta do domínio da educação sanitária. Não se pode jamais isolar o que se passa no interior da escola do conjunto de interesse e necessidades da família e da colectividade em geral.

A pedagogia moderna insiste *sobre a regra social da escola* e sublinha ao mesmo tempo a necessidade de tratar cada aluno em função da sua própria personalidade.

2.º — *Condições essenciais à personalidade e como o professor lhas pode favorecer:*

O ensino da higiene dá os mais eficazes resultados, desde que se tire partido de situações, interessando directamente os jovens, a fim de lhes fazerem compreender o porquê e como duma vida sã.

O ensino deve, portanto:

a) Ligar-se directamente às necessidades e interesses dos alunos, assim como das condições nos quais eles vivem.
b) Suscitar entre os alunos o desejo de adoptar o comportamento a seguir e adquirir os conhecimentos teóricos e práticos, nos quais eles se poderão inspirar na sua vida quotidiana na escola e fora dela.
c) Lançar as bases duma vida sã de futuro.

3.º — *Teorias de aprendizagem:*

À medida que se desenvolvem as possibilidades de instrução no mundo inteiro, incumbe às escolas preparar mestres que, em qualquer situação que se encontrem posteriormente colocados, possam contribuir para a melhoria da saúde, tanto individual como colectiva. Os temas de discussão em que se forma a preparação dos educadores em relação aos ensinamentos da higiene são os seguintes:

a) Aplicação prática dos princípios relativos à criança e à aquisição de conhecimentos.
b) Métodos de ensino sob o ponto de vista de educação sanitária.
c) Saber empregar as condições de meio imediato como base de educação sanitária, por exemplo:

— Utilizar as refeições escolares para ensinar a maneira de escolher e preparar os alimentos;
— Utilizar os terrenos e jardins da escola;
— Ensinar a costurar;
— Ensinar a purificação da água;
— Combate aos insectos;
— Evacuação de desperdícios e de lixos.

4.º — *Aprender a utilizar os serviços sanitários com base no ensino, por exemplo:*

a) Levar as crianças às consultas, dentista ou enfermeiro.
b) Preparar as crianças e seus pais com vista às vacinações.
c) Aplicar medidas de primeiros socorros.
d) Ter em dia *dossiers* sanitários e utilizá-los como base de educação sanitária.
e) Dar às crianças conselhos individuais sobre os problemas da saúde.
f) Organizar conferências, agrupando o professor, enfermeiros e pais.
g) Visitar os serviços sanitários.

5.º — *Informações de carácter relativo à saúde individual e colectiva:*

a) Os hábitos de higiene individual.
b) Adaptação individual e a saúde mental.
c) Higiene dentária.
d) A nutrição e a alimentação.
e) Perigo dos estimulantes e dos estupefacientes.
f) A preparação do indivíduo à vida da família e a função da alimentação.
g) Prevenção das doenças e dos acidentes.
h) A saúde da colectividade.
i) Organização de cursos sobre as ciências fundamentais destinadas a dar ao futuro mestre um aperfeiçoamento dos problemas sanitários modernos em matéria de biologia, química, psicologia, bacteriologia e sociologia.

6.º — *Programas sanitários destinados à colectividade:*

Como a escola pode ajudar os futuros mestres:

a) Participar eficazmente nos trabalhos das comissões ou conselhos sanitários.
b) Tomar parte nas actividades de instituições sanitárias oficiais ou colaborar nestas instituições.
c) Cooperar com os pais, os dirigentes da colectividade e todos os outros que se interessam pela saúde das crianças.
d) Colaborar com os responsáveis da educação, da colectividade e adquirir uma prática satisfazendo trabalho de grupo.

7.º — *Como elaborar e ministrar uma aula para educadores sanitários:*

Ter em vista os seguintes pontos:

a) Aparência e atitude dos alunos.
b) Voz — articular bem as palavras.

c) Vocabulário — construção de frases.
d) Originalidade na apresentação da lição.
e) Interesse e entusiasmo.
f) Autodomínio. Tacto.
g) Como apresentar o assunto para não ferir a susceptibilidade da pessoa.
h) Forma de apresentação das perguntas e como acolhem as respostas.
i) Critério demonstrado em relação aos assuntos que estão a ensinar.
j) Conhecimento do assunto que vai constituir a lição.
k) Forma como organizaram essa matéria.
l) Forma como ilustrar as lições. Apresentação no quadro preto.
m) Disciplina entre os alunos no decorrer da lição.
n) Quadro elucidativo de como se deve elaborar uma aula:

TÍTULO DA LIÇÃO

Classe ....

Objectivo da lição ....

I Princípio	*Para a 1.ª lição duma série:* Introdução geral à série. Introdução à 1.ª lição.	*Para as seguintes lições:* Resumo da última lição, definindo o objectivo. Introdução à nova lição, definindo o objectivo que a nova lição procura solucionar.
II Parte central	*Nova lição:* I — Título. II — Título. III — Título.	Nesta secção apresenta-se a matéria, isto é, todos os novos conhecimentos que irão solucionar o problema apresentado.
III Fim	*Resumo:* Dos principais pontos apresentados na parte central. *Aplicação dos novos conhecidos adquiridos.*	

8.º — *Métodos de ensino que podemos utilizar:*

a) *Perguntas e seus benefícios* — saber perguntar é uma arte.

Um bom professor distingue-se pela forma como interroga os seus alunos. As perguntas constituem uma das melhores formas de manter a actividade dos alunos.

O objectivo do professor, ao perguntar, é estimular o aluno; guiar o seu raciocínio e pensamento e obter a cooperação e a participação para a sua aula, evitando a indiferença

b) *Sugestões para o uso das perguntas* — atender a um determinado número de factores, sem os quais não obtêm os resultados desejados.

O *1.º factor* — que as perguntas sejam definidas e apresentadas duma forma clara.

O *2.º factor* — a considerar é a necessidade de dividir as perguntas a todo o curso, porque assim estimula o raciocínio a todos os alunos. É necessário dar um pouco de tempo aos alunos para pensarem. Só depois é que o aluno é indicado para responder.

O *3.º factor* — dividir as perguntas pelo curso.

O *4.º factor* — ajudar o aluno a responder.

O *5.º factor* — perguntas que não vão alterar a sequência clara e adequada da matéria. Quer dizer que as perguntas devem ser planeadas por escrito.

Ainda como métodos de ensino, podemos utilizar:

a) Quadro-preto.
b) Material ilustrativo: filmes, cartazes e prospectos.
c) Narração ou prelecção (casos de estudo).
d) Argumentação.
e) Experiência.
f) Demonstração (visitas de estudo e práticas nas diferentes instituições).
g) Exames: objectivos e descritivos.
h) Publicações pela imprensa, emissões e gravações.

i) Exemplos de conversas familiares: o educador sanitário terá de ensinar oportuna e momentâneamente, sem ferir susceptibilidades e usando da simpatia, que lhe deve ser inerente, lições sobre: higiene individual e colectiva, explicando sempre o porquê das coisas, descendo sempre até eles e atendendo às condições existentes. A melhor maneira é a conversar.

Por exemplo:

1.º — Porque deve lavar as mãos antes de comer.
2.º — Como e quantas vezes deve lavar os dentes.
3.º — Higiene da habitação.
4.º — Como arranjar e confeccionar (cozinhar) os alimentos.
5.º — Desinsectização.
6.º — Desinfecção.

Nota. — De preferência, todos os ensinamentos devem ser ministrados na língua nativa de cada região, para melhor compreensão.

THE DEMONSTRATION TECHNIQUE FOR STIMULATING TROPICAL DISEASE PROGRAMS [1]

D. J SCHLIESSMANN [2] [3]

Sanitary Engineer Director

Public health demonstration programs, as conducted throughout the world, have multiple objectives. They illustrate principles of control for solving an immediate public health problem, and they provide a facility for training personnel. A possibly more significant objective, however, is the development of awareness in the populace that something can be done to cure or prevent illness, thereby creating public suport and cooperation for a continuing program.

The purpose of this paper is to discuss the applicability of demonstration projects for the control of diarrheal disease through programs of environmental sanitation. While special attention is devoted to enteric disease, the principles of environmental demonstration projects also may be applied to the control of such tropical diseases as malaria, yellow fever, filariasis, bilharziasis, and many others.

THE DEMONSTRATION TECHNIQUE

A review of a number of successful public health demonstration projects indicates that the following factores were important: (1) there existed an acute problem; (2) technical personnel and information

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[1] For presentation before the Sixth International Congresses of Tropical Medicine and Malaria, September 5-13, 1958, in Lisbon, Portugal.

[2] Chief, State Aids Section, Technology Branch, Communicable Disease Center, Bureau of State Services, Public Health Service, U. S. Department of Health, Education and Welfare, 50 Seventh Street, N. E., Atlanta 23, Georgia.

[3] The author did not attend the meeting.

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were readily available to permit development and conduct of a scientifically sound program; (3) relatively favorable economic and logistic conditions existed or were provided from outside sources; (4) the program objectives were described and explained to the people at every opportunity; (5) citizen participation was obtained and provisions were made to employ and train local people to assure continuation of the project; (6) social, cultural, and religious customs were taken into consideration and control procedures were modified to avoid conflict with behavioristic pattern or practices of the group; and (7) the project had, or eventually obtained, the active support and cooperation of the community.

Within the United States considerable success has been experienced with Community Vector Control Demonstration Projects. The objective of these projects is to control vectors of public health importance by eliminating or modifying environmental conditions conducive to vector propagation. Insecticides are used only for supplementary control. In urban communities, environmental factors that contribute to vector production are associated with insanitary practices or conditions of refuse storage, collection, and disposal; poultry or other animal shelters; disposal of industrial or agricultural wastes; collection and disposal of excreta; housing; and residual water. In attempting to eliminate or modify these environmental factors, the project impinges on various agencies of municipal government other than the health department. For example, responsibility for refuse collection and disposal may be centered in the public works department; the problem of location of animal shelters in the zoning department; water, sewerage, and drainage in the engineering department; and total planning for housing and industrial development of the community in the planning department. This dispersal of responsibilities has necessitated leadership by the health department in the development of a coordinated program.

Such a project is undertaken by the United States Public Health Service cooperatively with a State and local health department. The Public Health Service provides a technical supervisor, nominal vector control equipment and training aids. The State Health Department assigns a staff member to provide assistance to the local project, to coordinate work within the State, to furnish educational posters and pamphlets, and to develop information for release to newspapers, radio, and television. The local health department employs a sanitarian to work full-time on the project. He becomes the *alter ego* of the project tecnical supervisor and subsequently assumes responsibility for the project. In addition, the local health department provides labor and material required for insecticidal or drainage work.

At the beginning of the two-year project the project supervisor and the local sanitarian make a complete sanitary survey of the city, prepare a comprehensive plan for improvement of sanitation, organize citizen participation groups, and carry out a generalized educational program. While total correction of environmental deficiencies is obviously impractical in a limited period of time, sufficient progress is achieved by the end of the first year to permit initiation of the second phase of the program. This involves active cooperation on the part of the State Health Department to publicize the program. Public officials in municipalities throughout the State are invited to the demonstration site, where the program is discussed and explained. The pride and enthusiasm of local municipal officials in their accomplishments and objective planning are communicated to visiting representatives.

In a midwestern State a project such as this was started in January 1957, and the promotional aspects of the program were begun in December of the same year. As a result of the interest of public officials who visited the demonstration city, 15 programs were started elsewhere in the State in the following six months in communities with populations ranging from less than 1,000 to more than 250,000. These satellite projects, located in different areas throughout the State, serve as demonstrations to adjacent communities.

The demonstrations have been successful because they have provided a solution to a complex problem, attendant publicity has kept the people informed, citizen participation has been received, local funds have been made available, and local personnel have been trained to assure continuation of the project. All of these factors must be considered in any plan to demonstrate control of tropical diseases.

DIARRHEAL DISEASE AND THE ENVIRONMENT

For the total control and prevention of diarrheal disease, the following environmental conditions are deemed essential (**1**) (1): provision of a saf, adequate water supplv under pressure within each home; (2) provision of sanitary methods for excreta disposal within each dwelling, and prevention of fecal pollution to ground and surface water supplies; (3) elimination of household and neighbourhood contamination of soil; (4) prevention of access of vectors, namely flies and snails, to human or animal feces; (5) housing with basic sanitary facilities, adequate space to prevent excessive crowding, and absence of rodents, vermin, and domestic animals; and (6) effective sanitary controls governing production, processing, storage, and consumption of milk and food.

The practical impossibility of achieving simultaneous improvements of these objectives, except possibly in localized areas, is apparent immediately. In general, it may be stated that where such ideal conditions exist, they were brought about gradually and by evolution in areas now characterized by a relatively high economic status and a correspondingly low incidence of diarrheal disease. Since such exemplary conditions were brought about gradually, it may be assumed that changes occurred primarily following the general improvement of the economic levels of a population. Improved economic status provided the means for step-by-step improvements in environment and resulted in modification of social or religious customs so that changing conditions were accepted.

If the above assumption is correct, then we are faced with an anachronism in our efforts to obtain area-wide control of diarrheal disease by environmental control in underdeveleped areas. The increasingly urgent concern of governments and health officials throughout the world for development of procedures to control diarrheal disease *now* precludes reliance on gradual and evolutionary improvements in economic status. The need is for the development of a positive and revolutionary program of prevention and control by environmental sanitation. The immediate result would be reflected in reductions in infant mortality, prolonged life expectancy, increased number of productive workers, and general betterment of the health and well-being of populations in underdeveloped areas. Improved environmental sanitation then becomes a cause rather than an effect of economic betterment.

Considering the theoretical requirements for environmental control of diarrheal disease, this «cart before the horse» approach may seem utterly impractical. However, consideration should be given to a step-by-step approach in which one environmental problem is taken up at a time. There is substantial evidence that modification of single environmental factors will result in reduced transmission and incidence of enteric diseases — a less desirable objective than complete control, but a more realistic one.

Studies in Texas (**2**) and Georgia (**3**) have demonstrated that reductions in incidence of shigellosis diarrheal disease were accomplished by means of fly control. These investigations were conducted in areas of moderate or low diarrheal disease, high fly populations, and ample opportunity for exposure of flies to fecal contamination. Fly control was obtained with chlorinated hydrocarbon insecticides. As the Georgia study progressed, failure to maintain fly control because flies developed resistance to the insecticides was accompanied by increased incidence of diarrheal disease. While these studies clearly showed that reduction in diarrheal disease could be effected by fly control with insecticides, the practicability of this method for the control of diarrheal disease is restricted.

The need for a bacteriologically safe water supply is self-evident. However, evidence has accrued which indicates that the *availability* of water adequate for personal hygiene is also of primary importance in reduction of diarrheal disease. Hollister *et al.* (**4**), in studies of migratory workers in California, reported that *Shigella* prevalence rates in children 10 years of age and under, who were living in dwellings with an inside water supply, were approximately one-half the rates of children living in dwellings with no convenient water supply. Similarly, for small urban communities in South Georgia, Stewart *et al.* (**5**), reported that *Shigella* infection rates varied inversely with the availability of water. These authors concluded that «the availability of water must be considered along with purity of water in any diarrheal disease program».

Subsequent studies in Kentucky (**6**), showed that the prevalence of diarrheal disease and *Shigella* and helminthic infections all were significantly related inversely to the availability of sanitary facilities. The authors concluded that, irrespective of etiologic or sociologic differences, enteric disease could be reduced significantly by selectively modifying the environment. While this hypothesis should be tested in a number of geographical areas and differing cultural groups, the accumulated data provide a basis for initiating objective and selective environmental control programs.

DEMONSTRATION PROJECTS IN TROPICAL DISEASE CONTROL

It is not intended to minimize the importance of «total» environmental control for reduction and prevention of enteric disease. However, we are concerned here primarily with consideration of the aplicability of demonstration projects for control of diarrheal disease in those areas of the tropics in which the majority of the people reside in villages without adequate water supplies. For many years, community improvement programs in tropical areas of Asia, Africa, and South America have largely emphasized development of a safe water supply. Possibly, because of economic considerations, less attention has been given to providing adequate distribution systems to make water increasingly available to the population. Water, in addition to being a basic physiological necessity for man, is essential for maintenance of personal hygiene. To satisfy man's *physiological* needs, it should be bacteriologically safe, palatible, and free of turbidity and color. To serve the function of its use in *personal hygiene,* adequate quantities must be conveniently accessible. In those areas where funds are not adequate to complete both treatment and distribution facilities, serious consideration should be given to the desirability of relaxing treatment plant design requirements, placing more reliance on chlorine residuals, and providing a more adequate distribution system.

In planning successful demonstration programs, public health officials should attempt to ally their activities with proposed agricultural or industrial projects designed to improve the general economic status of the population. The allied activity may involve plans for development of water resources for irrigation, power, or flood control. The dual objective of health and economic improvement should, wherever possible, be coordinated in planning programs in underdeveloped areas.

Another important factor in total planning for better environmental sanitation is cognizance that suggested improvements may be met with apathy, resentment, or lack of appreciation and acceptance due to conflicting social or religious customs. Therefore, the behavioristic patterns and practices of sociocultures differing from our own should be understood prior to initiation of environmental control programs. As stated by Paul (**7**): «If one wishes to help a community improve its health one must learn to think like the community».

In summary, demonstrations provide a ready means for illustrating techniques and methodology, provide an organization for concurrent training of native workers, and fulfill a needed health requirement with minimum expenditure of funds. It is reasonable to conclude that soundly planned and conducted environmental demonstration projects, can be applied to improve health and living conditions throughout the world.

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L'ÉDUCATION SANITAIRE EN ALGÉRIE: SON ÉVOLUTION — SES PROBLÈMES — DOCTRINES ET TECHNIQUE ADOPTÉES PAR LE CENTRE D'ALGER

A. C. LACROIX [1]

Directeur du Centre d'Education Sanitaire d'Alger

Dès le debut de la présence française en Algérie, le 14 juin 1830, les problèmes sanitaires se sont inscrits au premier plan des préoccupations du moment. Les arrivants se trouvaient en face d'un pays dont la population souffrait, entre autres choses, d'un état sanitaire des plus mauvais, frappée qu'elle était par les fleaux du paludisme, de la syphilis, des épidémies de typhus, de variole, des disettes permanentes et de famines périodiques. Les parties du sol les plus fertiles étaient recouvertes de marais meurtriers et d'immenses étendues de terre étaient incultes. Pas même deux millions d'êtres humains peuplaient cette contrée, dont la situation démographique était, à tout le moins, stationnaire.

La médecine était exercée par des empiriques et des charlatans. Il n'existait pas de formations hospitalières.

L'hygiène tirait ses directives des précepts du Coran, qui, par ailleurs certainement valables, avaient dans la pratique perdu toute efficacité, deformés qu'ils étaient par leurs interprétations et leurs applications traditionnelles, au point d'aller souvent à l'encontre des buts recherchés.

Ce pays, qui avait connu des heures de grandeur et de prospérité, parcourait un trajet de la ligne de son histoire dans lequel s'inscrivaient la décadence, le croupissement et la misère. L'action de la France inflichit brusquement cette ligne en la dirigeant vers les sommets des idéals humanitaires les plus purs.

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[1] The author did not attend the meeting.

Dès 1831 l'Hôpital de la Salpetrière est créé à Alger. En 1844 il y a déjà 32 hôpitaux. En 1845 le nombre est de 38 et il passe à 75 en 1927, pour atteindre 139 en 1956.

Parallèlement, l'action sanitaire itinérante se développe: en 1844 les médecins militaires des Centres Médicaux des Bureaux Arabes effectuent de nombreuses tournés parmi les populations. En 1853 le cadre des médecins de colonisation est créé, avec 27 postes. En 1875 il y en a 59. En 1940 il sont 912. Actuellement: 57 médecins inspecteurs, 166 médecins de l'assistance medico-sociale, auxquels s'ajoutent 100 médecins contractuels à temps plein et 140 médecins conventionnés à «temps partiel».

Cette action thérapeuthique ne va pas sans l'éducation sanitaire des populations au contacte. Cependant, les efforts ne sont pas exactement recompensés d'un plein effect: les populations restent assujetties aux traditions, aux coutumes, au fatalisme.

En 1927 un programme raisonné et précis est établi en vue de l'organisation de la médecine préventive et de campagnes méthodiques d'éducation populaire en matière d'hygiène. L'Office Algérien de Médecine Préventive et d'Hygiène utilise une camionnette authomobile avec groupe électrogène pour projection cinématographique. Deux médecins, dont un Musulman, sont désignés pour commenter les films. Des disques phonographiques en langue arabe sont édités. L'Institut Pasteur d'Alger, fondé en 1894, publie des tracts sur le paludisme et édite une brochure illustrée de schémas — *Kitab ec-ciha* (le livre de la santé).

La seconde guerre mondiale contraint à une pause qui se prolonge jusqu'à la fin de 1945.

Cepandant le terrain parcouru peut se mesurer objectivement, d'une part, par l'accroissement démographique — 2 946 067 habitants en 1956, 8 681 785 en 1948 — et, d'autre part, par la quasi disparition de la syphilis, la suppression des épidémies de choléra, de variole, de typhus; la regression considérable du paludisme, de la mortalité maternelle et infantile; de la disparition des famines.

Le bilan de l'action humanitaire de la France est extraordinaire, et pourtant il n'est pas celui désiré: la tuberculose poursuit des ravages, favorisée dans son extension par l'ignorance des populations menacées — le paludisme résiste encore — le trachome reste le fleau dominant. L'oeuvre éducative doit se poursuivre, en accentuant son efficience.

Pendant les dix années qui viennent de s'écouler, l'éducation des populations a particulièrement retenu l'attention d'organismes mondiaux: l'UNESCO et l'OMS. Nombreuses ont été les réunions des groupes d'experts de congrés internationaux. Des campagnes ont été organisées. De cet ensemble de travaux se dégagent des doctrines et des méthodes. Et en conclusion, des guides et des schémas ont été élaborés en vue de la mise en pratique de l'éducation sanitaire. Mais il s'agit là de thèmes directeurs qui offrent les inconvénients du planisme parfait. Dans le concret, c'est souvent le moindre mal qui est le premier pas nécessaire et valable dans la voie du bien. Il n'est pas de plan préfabriqué qui ne nécessite des adaptations souvent considérables et toujours l'efficience est d'autant meilleur que l'action est coulée dans le moule des réalités du lieu d'application.

Les aspects de l'éducation sanitaire en Algérie sont marqués du triple sceau:

— Celui des croyances, des coutumes, des traditions faussement imputés à la religion, mais ayant acquis la puissance de dogmes;

— Celui de la dissémination des populations rurales et nomades;

— Enfin, celui du sous-développement d'une fraction importante de la masse.

Nous n'ignorons rien des efforts entrepris dans le cadre de l'éducation de base. Mais il faut satisfaire à l'impératif: agir immédiatement animé par la volunté d'obtenir un rendement valable. Cette action immédiate ne doit ni entraver ni être entravée par les autres actions menées parallèlement et conjointement, actions dont les résultats ne deviendront sensibles qu'à une échéance plus ou moins lointaine.

C'est dans cet esprit que depuis 1946 nous avons conçu et mis en pratique, dans les limites des moyens financiers mis à notre disposition, une technique d'éducation sanitaire par le film cinématographique.

Voici la dernière production du Centre Regional d'Éducation Sanitaire — *protegeons nos yeux*, qui illustre les conception que nous jugeons valables pour l'Algérie.

Le film, en couleurs, a été pris dans le cadre même des milieux à éduquer — les personnages sont des habitants des villages.

Le scénario a été conçu en accord avec le commentaire etabli en puisant dans les textes coraniques et en poursuivant deux objectifs: l'un, attirer l'attention sur les fautes d'hygiène à éviter; l'autre, de mettre en évidence le traitement simple commun à toutes les affections oculaires, le *leitmotiv* «des gouttes dans les yeux».

Le commentaire, dont est ci-après jointe la traduction française, ne comporte aucun terme médical. Les idées exprimées sont aussi simple que possible; elles sont exactes, c'est la vérité, mais pas toute la vérité, seulement l'essentiel de la vérité.

La bande filmée est muette.

Elle doit être commentée synchroniquement:

- — soit par un commentateur autant que possible équipé d'un microphone,
- — soit à l'aide d'une bande d'accompagnement magnétique se déroulant synchroniquement sur un magnétophone.

Cette technique offre un certain nombre d'avantages:

1° Celui de permettre l'utilisation de la même bande au profit de collectivités de langues différentes (français, anglais, patois arabes, kabyles, etc.); c'est le commentateur du lieu, ou la bande magnétophone adéquate d'accompagnement, qui assure l'adaptation au public;

2° Celui de réduire considérablement le prix de revient des bandes filmées par la suppression de la bande sonore.

Or l'élément important de son efficience est la grande diffusion de l'éducation sanitaire. Cette diffusion doit être facilitée par l'abondance des bobines éducatives et leur renouvellement fréquent.

Ce dernier point constitue un impératif. Soulignons que:

- — les thèmes d'éducation sanitaire sont toujours les mêmes;
- — le public à éduquer reste toujours le même, à la différence prêt de l'apport des générations nouvelles;

— la répétition est un des facteurs d'efficacité de l'éducation.

Il est donc nécessaire de renouveler fréquemment le matériel éducatif: un film ne doit pas être utilisé pas plus de deux ans, à peine d'engendrer le désintéressement et l'éloignement du public. Ainsi se trouve réalisé le «type» de l'éducation sanitaire evolutif, mouvant, plastique, vivant.

Au surplus, cette technique devient hautement valable dans l'application de certaines directives. La doctrine du Centre Regional d'Éducation Sanitaire d'Alger s'inspire des principes suivants:

1° Les éducateurs sanitaires sont divisibles en deux catégories:

— l'une, celle des éducateurs professionnels, peu nombreux, et dont le rôle est comparable à celui des chefs d'orchestre;

— l'autre, celle des éducateurs occasionnels, qui sont légion, et dont le rôle est celui d'exécutants volontaires dressés pour une action déterminée;

2° L'éducation sanitaire est donnée à «postes fixes» par les commentateurs occasionnels habitants du lieu. C'est le matériel éducatif, en particulier les films, qui est itinérant, allant d'un poste à un autre suivant un circuit déterminé.

Cette technique permet de répondre à tous ces impératifs, parce que son prix de revient est minime au régard de l'étendue de ses possibilités de diffusion.

Voici, en francs français, le prix de revient du film 16 mm, Kodachrom, *Protegeons nos yeux*, realisé par le Centre Regional d'Éducation Sanitaire d'Alger, de 12 minutes de durée:

1° tournage et montage du film	628 924
2° prix des copies, à l'unité :	
pour 10 copies	37 642
pour 20 copies	33 184
3° soit prix de revient par film, à l'unité :	
10 copies	100 534
20 copies	65 076
50 copies	49 720

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Le Centre d'Alger a créé une école de commentateurs, dans laquelle elle forme les éducateurs occasionnels.

Il assure la distribution des films éducatifs.

Mais, également, il édite deux sortes de semi-périodiques:

1° Les Cahiers Algériens de la Santé, adressés aux médecins de la santé, aux écoles d'infirmiers et infirmières; à toute les écoles primaires;

2° Les cahiers du Centre Regional d'Éducation Sanitaire envoyés aux éducateurs sanitaires, et devant servir d'organe d'information et de liaison.

Enfin, il diffuse des causeries radiophoniques en langues française et autochtones, sur les chaînes de la Radiodiffusion Française en Algérie — et il a réalisé une vitrine-exposition parlante dans un des lieux les plus passant d'Alger.

Pour terminer, donnons un exemple de l'application de la doctrine et de la technique exposées ci-dessus, par le Service de Santé de l'Armée en Algérie: les films d'éducation sanitaire, accompagnés de la bande magnétophone correspondante (matériel fourni par le Centre d'Alger), sont régulièrement en circulation parmi la troupe, mais également parmi les formations para-militaires. En 4 mois, 145 séances ont été données, auxquelles ont assisté 14 mille soldats et également la population civile; leur contacte.

Mais l'exemple spectaculaire, demonstratif de la plasticité des moyens, a été donné par le lieutenant *Armandi,* chargé de la direction d'un Bureau d'Action Psychologique et Social de l'Armée, qui, envoyé à Malouza le lendemain même des massacres tragiques qui ont ensanglanté cette regicn, a effectué une projection d'éducation sanitaire à Beni Ihman devant 250 personnes, et aux Ouleds Taïr au camps des personnes regroupées devant un millier de spectateurs.

L'abîme est incommensurable, qui sépare les ignorants idéologues et dissertateurs des réalités de l'action généreuse poursuivie par la France en Algérie, avec la ferme détermination de conduire les populations dont elle a pris la charge le 14 juin 1830, vers les sommets de la civilisation et la pleine jouissance de ses bienfaits.

Il va sans dire que le Centre d'Éducation Sanitaire d'Alger répondra avec empressement à toutes les demandes de renseignements qui lui seront adressées: Laboratoire d'Hygiène — Faculté de Médecine — *Alger.*

CONDIÇÕES SOCIOECONÓMICAS E PREVALÊNCIA DE PARASITOSES EM ALGUMAS ÁREAS DO NORDESTE DO BRASIL

DR. MARCELLO COELHO [1]

O Nordeste do Brasil é uma região composta por 8 estados, compreendendo uma área de 12 240 496 km², o que representa 14,33% da área total do país. É uma região com características fisiográficas e humanas bem determinadas. Apresenta território semiárido, em grande parte sujeito ao fenómeno de secas periódicas. Apenas estreita faixa de terras costeiras e algumas zonas férteis, geralmente próximas às serras, perfazendo, em conjunto, pouco mais de 132 000 km², apresentam-se cobertas de matas. O revestimento florístico do restante da região, cerca de 67 %, é formado por catingas. No Nordeste vivem aproximadamente 19 000 000 de habitantes, 31 % da população brasileira. Do ponto de vista económico, a região apresenta todas as características gerais das zonas subdesenvolvidas. Curta duração de vida (25 a 30 anos, em média), com aproximadamente 77 % da população se dedicando à agricultura, enquanto apenas 6,71 e 4,03 %, respectivamente, à indústria e ao comércio. A renda anual *per capita* é baixíssima. Nos biénios de 953-54 foi de Cr $3 000 000, contra 7 300,00 para o total do país. Em alguns estados da região é apenas de cerca de Cr $2 000,00 — 7 vezes inferior à do estado de São Paulo. É ainda 3 vezes mais elevada nos centros urbanos que no meio rural. O Nordeste contribui apenas com 13,11 % da renda nacional, apesar de apresentar quase a terça parte dos habitantes do país. O desequilíbrio económico na região é alarmante. Um só estado, o de Pernambuco, contribui com 24,95 % da renda regional. As riquezas se concentram, sobretudo, nos poucos centros industriais, geralmente situados nas regiões costeiras, onde uma pequena parcela da população, cerca de 5 %, percebe quase 40 % da renda global da região. O comércio se apresenta como actividade mais importante que a indústria. O produto agrícola de mais valor e de maior extensão cultural da região — o açúcar — é um produto de baixo preço. Com uma população activa remunerada de apenas 31 % da população global, a região se constitui actualmente em área de emigração e de fome crónica. A mortalidade infantil é alarmante — de 300 por 1 000 crianças, em algumas zonas.

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[1] The author did not attend the meeting.

As condições económicas citadas têm como consequência o estado de vida extremamente primitivo de grande parte dos habitantes da região, sobretudo nas extensas áreas rurais. O destino adequado dos dejectos, o uso do calçado, de água tratada, a alimentação suficiente, quantitativa e qualitativamente, atingem apenas ínfima percentagem da população regional. As condições são extremamente favoráveis à prevalência de parasitoses. A esquistossomose mansónica incide em todas as áreas onde são encontrados moluscos vectores, com índices algumas vezes aproximados a 100 %. É prevalente graças à convergência de vários fatores favoráveis. Os criadouros de moluscos são, em numerosas áreas, a única fonte de água para os trabalhos domésticos, para o banho, para a lavoura. É generalizado o hábito de usar estas mesmas fontes para destino de dejectos humanos. A ancilostomose, com incidência de 77,0 %, é assinalada em todos os estados da região. O destino inadequado dos dejectos e a falta do uso de calçado podem ser responsabilizados por esta grande incidência. O calçado, para grande parte da população, é ainda objecto de luxo, inacessível aos orçamentos familiares, que mal possibilitam a subsistência, em condições verdadeiramente sub-humanas. As outras verminoses do tubo digestivo, sobretudo a ascaridiose e a tricocefalose, são encontradas em, pràticamente, todos os habitantes da zona rural e em grande proporção dos habitantes das cidades. A tuberculose e a sífilis são assinaladas frequentemente, sobretudo nos centros urbanos. O tracoma, a doença-de-Chagas e as leishmanioses, cutâneo-mucosa e kalazar, incidem nas zonas rurais, enquanto as doenças gastrintestinais e as carências nutritivas são encontradas em todo o meio rural e, mesmo, em grande parte da população das cidades. Na região, até o presente, foram apenas controladas as doenças pestilenciais, a malária e a bouba, graças à acção de serviços federais, ajudados algumas vezes por organismos internacionais.

A análise conjunta dos factores sanitários e socioeconómicos da região indica a pequena possibilidade de êxito de qualquer trabalho educativo, visando a profilaxia destas parasitoses, sem a modificação da conjuntura económica regional. Especialmente para determinadas endemias, como a esquistossomose, as verminoses, as doenças carenciais, esta necessidade apresenta-se evidente. Experiências realizadas pelo Departamento Nacional de Endemias Rurais, em vários municípios da região, têm demonstrado serem ineficientes as medidas educativas em núcleos de população não assistidos econòmicamente. Por outro lado, tem sido observado, em alguns municípios mais prósperos, que são ineficazes como medidas profilácticas contra muitas endemias, sobretudo as de origem parasitária, medidas apenas tendentes à elevação do nível económico, não acompanhadas de trabalho de educação geral e sanitária, visando a modificação de hábitos estabelecidos, responsáveis pela prevalência destas parasitoses. Desta forma, embora a eliminação das causas do subdesenvolvimento e a consequente elevação do nível social e económico das populações, sobretudo na zona rural, possibilite as condições adequadas, para a modificação do quadro de prevalência das várias endemias, acredita-se que sòmente com o uso simultâneo de medidas educativas, sobretudo de educação sanitária, será possível o *contrôle* e a erradicação destas mesmas endemias.

GENERAL DISCUSSION

Dr. L. Roberts (Ghana) — I should define Tropical Medicine and Hygiene as a correlation of 3 p's — people, places and parasites. To achieve success you have to know this and always keep this in mind.

The doctor practising in the tropics must be a socialist as much as a scientist.

Tropical peoples are interested in their children, their food, their growth and their happiness. I suggest too that they have a fund of common sense and a lot of good humour.

Thus there is nothing that we cannot do. As doctors, we should win their suport, encourage self help and capitalise their assets.

The spoken word, films as well as practical demonstrations will appeal powerfully to them.

We in Ghana make great use of the Department of Social Welfare and Community Development. A programme of health education in the villages and rural areas is widely carried out, and I may add with great popularity and growing success too.

Health inspectors, health visitors and trained nurses and midwives also help to popularize this work.

Dr.ª Deolinda Martins (Portugal) — Permito-me sugerir que em qualquer país onde se inicie um curso de educadores de saúde não se pretende só fazer *profissionais* ou *técnicos*, mas, e *sobretudo*, introduzir o espírito de educação sanitária em todos os serviços públicos e populares em geral.

Não se podem fazer programas de educação sanitária, com resultados positivos, só com educadores sanitários, mas com a modificação de atitudes da população responsável em geral, pela preparação dentro do espírito da educação sanitária de todo e qualquer servidor público.

Os cursos devem ter menos informação médica e mais informação social, relações humanas, etnografias e mais trabalho de campo do que aulas teóricas.

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O curso deve ter apenas a duração máxima de um ano, se for um curso complementar.

PROF. HASSELMAN — I strongly endorse the recomendation to approach the population through their head-men, elders, et *genus omne*, to instill basic concepts and targets of the particular health-campaign into the people. It is most helpful and essential to the success to win the confidence and understanding of these respected men and women in a community who in their turn will translate into the undestable language and explanations to their families and dependents the objectives and advantages of that particular health project, whether against yaws, malaria, tuberculosis, schistosomiasis, trachoma, infant mortality, etcetera. We in the Philippines could largely count on the excellent Philippine Health Service personnel and nurses, and in Indonesia the previous Dutch Colonial Administration had succeeded in establishing reliable census figures which were still available, and of excellent value in 1950-1951 to survey and assemble the people in the different villages for examining in the control campaings against yaws and other tropical maladies. In Thailand we received in our World Health Organization's field control measures most valuable cooperation from the Buddist priests once they were convicingly informed by us about the objectives and foreseable results of our control work. All these educational efforts in the planning stage of the field control measures and during the initial phases vastly enhanced our protean labors in the field. On the other hand, a few isolated instances of attempts to attenuate health campaign objectives by raising religious issues might tend to defeat all earnest efforts. E. g. in some highly orthodox Mohammedan districts inoculations were discriminately labelled «Sentuki serani» i. e. Christian injection, or, worse, the scar from smallpox vaccination sneeringly called «Tjap Company» i. e. the mark of the Company, inasmuch as the big plantation compagnies had painstakingly vaccinated their labor population and families against smallpox. To forestall such discriminations and prejudices, utmost educational and preventive information of the populace, and here best by adequate and tactful approach through their trusted elders, teachers, councillors and leaders is imperative as prophylaxis against misunderstanding and misconceptions and essential to reach the objectives of any public health campaign with a minimum of failure and waste of time, money and effort.

SUBSECTION 2

HOUSING AND INDUSTRIAL HYGIENE

1ST SESSION: **Housing** — *Friday, September, 12th — 9:00 to 10:30 a.m. Room B*

The meeting was held under the Chairmanship of Dr. Young-Choon Lee who took the chair in the absence of the appointed Chairman Dr. A. H. Baldwin.

The Rapporteur was Dr. Ronald K. Macpherson and the Secretary was Dr. Mário A. de Mendonça e Silva.

The following papers were presented and discussed:

SPECIAL PROBLEMS OF LARGE SCALE URBAN HOUSING IN SINGAPORE

J. M. FRASER, C.B.E., E.D., F.R.I.B.A., M.T.P.I., M.R.S.H.

STATISTICS

Area	224–5 sq. miles	Public roads . . .	480 miles
Equator	85 miles.	Traffic — Cars . .	70,000
Rainfall	95 inches	Bicycles	230,000
Temperature . .	70° to 90° F.	Shipping.	60 million tons per annum

Population	$1\ ^1/_2$ million.
Natural Increase Rate	4 $^0/_0$ per annum

U. S. $ 1 = $ 3 Singapore.

Singapore Improvement Trust has built 18,000 dwellings, shops and markets since 1947 at a cost of over $ 110 million.

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 674-691, 1959

FRASER, J. M. — *Urban housing in Singapore*

INTRODUCTION

Singapore has been chosen specifically as the subject for this paper as the author's experience for the past 31 years has been with the Singapore Improvement Trust. Obviously the problems in other countries in the tropics will differ considerably, not only because of variation in climate but also in relation to population, geography, race, religion; also the main occupations of the people and the economic circumstances which affect them. The conditions affecting housing in Singapore are very different to those in Hong Kong and likewise different from the mainland of Malaya. Hong Kong is an island with similar overcrowding and space problems but its climate is different and so are its political and economic conditions. Similiar problems arise but the solutions to these problems are not necessarily the same. In Malaya land is a much less important factor than in Singapore as it is more plentiful, and timber is a more suitable material as it is in better supply. Single storey housing is an economic proposition which it is not in Singapore. It is therefore difficult to generalise and not always wise to attempt to meet one country's problems with another's solutions.

Problems in large scale urban housing anywhere can be divided into three main headings, e. g. *Social, Structural* and *Economic*. These three cannot always be kept apart and indeed often dovetail into one another to create a dilemma for the Housing Authority that defies solution. I shall try in this paper to keep them separate but they will inevitably overlap.

SOCIAL

The main problem in Singapore is the high rate of population increase — 4 % per annum by natural causes — probably the world's highest! Medical science, a remarkably healthy climate and the complete elimination of malaria has contributed to make the population a young and healthy one with an average age of twenty and a death rate of about eight per thousand. Out of a population of a million and a half, more than 250,000 live in slum conditions not necessarily because they cannot afford something better but

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because better housing is not available. In the past decade Singapore has enjoyed the highest standard of living in South East Asia but the population as a whole are keener to run cars and buy radio sets than to save money to buy their own house. There are of course exceptions, but there is a natural reluctance to pay a reasonable rent for suitable housing accommodation and there has been much political agitation during the past few years for lower public rents and heavier Government subsidies. This is of course a popular political stamping ground but the result has been that they have nearly killed the goose that laid the golden eggs, and the prospect of large annual contributions to subsidise public housing is now facing a country which in the near future will be self-governed if not self-supporting.

The task in Singapore is to house in satisfactory conditions an annual population increase of nearly 60,000 and to try prevent the creation of further slums. Slums are created in many ways but the surest and quickest way is to allow overcrowding. Good dwellings can deteriorate rapidly if overcrowding and slum conditions prevail and no matter how good the buildings are, god tenancy management is of the utmost importance.

Chinese account for 80 % of the population of Singapore but more than 90 % of the population of 20,000 dwellings built and let by the S.I.T. are occupied by Chinese. In general they are good tenants who take a pride in their homes and are clean and tidy as far as their own doorstep. Unfortunately public spiritedness is not their long suit and they depend on the Housing Authority for maintaining the common passages, staircases and open spaces in and around the buildings in a clean and tidy state. Hence a large staff of estate labourers are employed for day-to-day maintenance and this costs the Trust about $ 3 per month per dwelling. Although the Chinese predominate, Malaysians, Indians, Pakistanis, Arabs, Jews, Eurasians, etc., generally mix happily together in S.I.T. housing and in Singapore generally.

Later in the paper I have discussed in more detail some of the problems of Estate Management but on the whole it is a source of great satisfaction to see families come from appalling slum areas and settle down in an estate of multi-storey flats in decent selfcontained roomy dwellings where the children can have some freedom to laugh and play and the housewife can take a pride in her own kitchen and lay new linoleum on her cement floor in the knowledge that she has at last got a secure home.

STRUCTURAL

The Trust commenced its post-war building programme in 1946 and considerable research and experiment in economical structural design has been carried out in the past 12 years. In 1946 after the liberation of Singapore there was a tremendous housing famine and ways and means had to be found to start up a housing programme in spite of the high cost of materials and labour. The authorities in spite of agitation wisely rejected proposals for very temporary wood and attap house which would have wasted land and been a source of worry and expense for their short and uneconomical life. The architects of the Trust evolved a type of reinforced post and panel single storey dwelling of units fabricated on the site and about 600 of this type were built in the course of the next three postwar years. These are two room living units of 442 sq. ft. with enclosed yard of 150 sq. ft. in the rear, and cost $ 3000 (Straits) = U.S. $ 1000 each including site works, access roads and drains. They were let at $ 20 a month and were deservedly popular. They are not regarded as permanent having a useful life of say 40 years and as they are built at densities of about 20 houses per acre, are uneconomical in land. The layout is attractive and there is plenty of fresh air and playing space. They are ideal family houses and suitable for housing at densities of up to 200 persons per acre.

THE MASTER PLAN

A special committee prepared a Housing Report in Singapore in 1947 and this report clearly showed that the most important and immediate requirement was for a Master Plan to be prepared so that the available land on the island could be used to the best advantage.

In 1952 a team was formed to carry out a diagnostic survey and prepare a Master Plan, with Sir George Pepler as Planning

Adviser to the Government. The Plan was completed in 1955, published in 1956 and a public inquiry held in that year. The Plan as amended has been accepted by the Government of Singapore and legislation is being put through for its implementation.

This plan provides for residential densities of up to 600 persons per acre in the central areas and this means over 100 dwellings per acre. The S.I.T. has carried out a number of large central area schemes at this density and with good internal planning this type of housing can be healthy and attractive. The bulk of the housing carried out since 1948 has been in multi-storied dwellings ranging from three to fourteen stories. From the point of view of land, roads and services, single storey housing is uneconomical and is now only adopted for rural or semi-rural sites. In the central areas the cost of land makes multi-storey housing imperative and the desire of the population to live near their work justifies the policy of providing housing in the central areas.

It will be seen therefore that the problems of planning, finance and construction overlap and solutions have to interlock.

PLANNING

The S.I.T. carries out day-to-day planning control over the whole island within the framework of the Master Plan and under the mandate of a most powerful clause in the Singapore Improvement Ordinance which states: «No person shall, without the written permission of the Board, put to a new use any land or building provided that the use of land for agricultural purpose shall not be deemed to be a new use of such land».

HIGH DENSITIES

This very wide power creates in itself a special problem in that only a limited amount of land is designated for urban development and housing has to take its share. The shortage of good housing land is one of the major problems facing the housing authority and that which can be made available has to be developed at high density. Fortunately the Chinese are a gregarious people and do not mind high densities. By Western standards more than half the population of Singapore live in overcrowded conditions and the slum areas in Chinatown have residential densities up to 1500 persons per acre. Rehousing of these people in multi-storied blocks of flats can be quite satisfactory at densities of 500 per acre and more, and the Master Plan allows for densities up to 600 per acre. The average family unit throughout S.I.T. housing is seven and this is about the average size of the Chinese household. It is made up of husband, wife, three children, mother-in-law and cousin (or some other alleged relative). In cases of rehousing the families are generaly smaller and in slum clearance areas when reacommodation is guaranteed, there is a large proportion of single persons or couples which makes rehousing very difficult. Bachelors are rehoused usually at two and three per room of 140 sq. ft. and small families are asked to share a two-roomed flat of 400 sq. ft. including kitchen, bath and W.C. regarded as being suitable for eight persons. This is not a satisfactory solution as two housewives using the same kitchen never really works. The only economical solution to the small family is the single room tenement with common kitchen, wash place and lavatories. The snag here is that unless water is carefully rationed the taps run all day and water is wasted. Recovery by service charge is no real answer as no one is ever satisfied. It is a curious human trait that when people do not pay directly for a service they have no regard for economy. They regard the service as free and bitterly resent having to pay for it indirectly.

COMMON SERVICES

It has therefore been the policy of the S.I.T. that wherever possible tenants will pay directly for the services they use. This can only be done by metering these services, i. e. water, gas and electricity, and the provision of separate kitchens, baths and W.C.s for each flat. This costs more money initially but is well worth while in the end. It shows a long term saving in supervision and maintenance.

Communal living in tenements under reasonable conditions is generally acceptable to the family which has never enjoyed anything better than a slum dwelling, e. g. the common kitchen for twelve families work quite well if each one has his own pots and locker and there is sufficient space. It cannot be compared, however, to the amenity of a private kitchen.

OVERCROWDING CONDITIONS

The only overcrowding standard laid down in the Singapore Municipal Ordinance is a minimum of 350 cub. ft. per person. This was laid down 45 years ago but is still regarded as reasonable. With 8 ft. ceilings it allows 40 sq. ft. floor space per person.

Large numbers of families in the central areas live in conditions of constant overcrowding and the S.I.T. housing register points system awards overcrowding priority in the following scale:

	Points award
Floor space per person over 30 sq. ft.	0
» » » » 24–30 sq. ft.	1
» » » » 19–24 sq. ft.	2
» » » » 15–19 sq. ft.	3
» » » » 12–15 sq. ft.	4
» » » » 10–12 sq. ft.	5
» » » » under 10 sq. ft.	6

Other factors are:

(*a*) Family unit	$1/2$ point per person with maximum of 3 points.
(*b*) New dependents & divided family	$1/4$ point per person.
(*c*) T. B. incidence	Maximum of 3 points.
(*d*) Eviction by Court Order for development	6 points.

HOUSING MANAGEMENT

From the above table it will be seen that priority in allocation of accommodation available is considerably influenced by the degree of overcrowding and at the time of writing ten points are required to obtain an allocation. Investigation officers after the first inspection make spot checks to verify if the information obtained is correct and the family is still living under the conditions as recorded, before allocation of accommodation. It is very often found that members of the household are «borrowed» for the purpose of boosting the «census» and never actually up residence in the flat when allocated. It is the job of the Lady Housing Visitor to see that the right family and the right people (as recorded in the tenancy agreement) are living in the new accomodation.

The Lady Housing Visitor (each in charge of 1200 properties) is a well educated person, over 30 years of age and usually married. She has to be able to speak a few dialects and acts as a kind of welfare adviser, detective and inspector rolled into one. She very often has to teach the family how to live in their new home and how to use the amenities provided in the proper way. She has to explain the tenancy rules and see that they are kept. She advises the tenants in many matters and by instinct and female intuition usually knows when something is wrong. She does not collect rent. Tenants are expected to pay their rents monthly in advance at the head office or the estate rent collection centre, and male rent collectors are engaged only in the collection of rents in arrear. District Estate Offices provide facilities for Estate supervision, maintenance and rent collection as well as headquarters for the outside staff.

The problems of Estate Management are mainly human problems and have to be dealt with in a humane manner. When there is full employment the problem of rent arrears is not a serious one but there must be constant vigilance by the financial officers. Distress action is taken at an early stage in an arrears case and this has often to be followed by notice to quit and court order for possession. In hopeless cases the tenant often absconds before eviction but the proportion of write-offs is small compared with the rent roll. Tenants will take advantage of a sympathetic administration and firm action must go hand in hand with fair dealing. The Social Welfare Department works in close liaison with the Trust and helps wherever possible with difficult cases.

HOUSING REGISTER

A housing register is kept up to date and at the time of writing had approximately 13,500 families representing approximately 100,000 people on lit. This does not reflect the total housing need in Singapore as the basic qualifications for registration are:

1) Singapore citizens of not less than 21 years of age.
2) Resident in colony for 5 years.
3) Not less than five in family unit.
4) Income limit of $ 600 per month.

In addition there is a rehousing register with 3,500 families who are living in house scheduled for demolition for town improvement, slum clearance or redevelopment schemes, and there are always fire victims on the list. Priority is given in accordance with the urgency of the particular cases on this list and a balance has to be struck between rehousing from this list and the public register.

In 1958 approximately 5,000 houses will become available for occupation and at this rate the register should be cleared in three years, but names are added to the register as fast as houses become available and a large becklog of slum clearance has to be dealt with in addition.

SLUM CLEARANCE

In addition to an estimated 250,000 people who are living in overcrowded conditions in the central areas, a further 175,000 people live in plank and attap Kampongs within the Municipal Area. The conditions in the Kampongs, although not so crowded as central are shophouses, are most insanitary. The houses are built in almost continuous rows along unpaved alleyways with no proper drainage or sanitation. A few standpipes here and there deliver water and night-soil is collected from bucket lavatories. There is constant risk of fire and disease is repelled only by the sun and rain. Improvements to drainage, access and water supply are as much as can be done and in spite of these conditions the population keeps reasonably healthy. The problem of clearing these areas and rehousing the people in them is virtually insoluble under the present economy which is not expanding in proportion to the great annual increase in population. Individual families here and there are rehoused when their turn comes on the register but as soon as their rooms are vacated they are reoccupied by some other family and the slum conditions continue.

Clearance schemes to provide for public and other development are carried out according to programme but it is virtually impossible to satisfy all classes of people who live and work in these areas. Although rents of public housing are heavily subsidised they are still beyond the capacity of a number of the poorest families who have to rely on public assistance for a roof over their heads, Chief Tenants and similar racketeers prey on the unfortunate circumstances of these people and extort ridiculous sums for «tea money» and «protection money» from the uneducated. There are always those who are ready to exploit the misfortunes of the poor and illiterate and they are the chief bugbears of the housing authorities. A large clearance scheme for the rehousing of over 500 families in an area notorious for many years has been help up indefinitely although housing accommodation is available due to political activities of Chief Tenants. In spite of these difficulties slum areas are being cleared and dwellings have been erected in such areas over the past ten years.

ECONOMICS

Rents of post-war Trust houses range from $ 22-$ 100 p.m. Up to 1956 rents were not subsidised by Government but the Trust's housing accounts ran at a loss.

Housing loans totalling 140 million dollars have been approved by the Singapore Government since 1948 and 30 million dollars per annum will be required to complete the current 5-year programme of 20,000 houses by 1962. It is proposed that a large part of this five year programme will be devoted to the rehousing of families dishoused from slum clearance areas.

It has been agreed that the minimum requirements for a family is a two-room self contained flat with separate kitchen, bathroom and W.C. with a total area of approximately 400 sq. ft. This flat is regarded as being suitable for a family of two adults and six children and conforms with the space standard of 350 cubic feet per person in the Municipal Ordinance.

Average cost of this flat in multistoried building $5,000
Plus cost of piling up to $1,000
With repayment of loan @ 5 $^1/_4$ % interest over 60 years.
Cost of land at a nominal charge of $ 1 per sq. ft.
City assessment at 34 % gross rent.
Supervision and maintenance charges of $220 per unit per annum including cost of the Housing Register.
Redecoration and repairs at $ 60 per unit per annum.

The economic rent works out at approximately $70 a month. Government has decided that the standard rent of these flats should be $35 a month so that they carry a 50 % subsidy. This rent amounts to one sixth of a monthly income of $210, which is an average for the lower income group. Five thousand housed at a subsidy of $35 per house per month amounts to an annual subsidy of $2,100,000 and if an annual rate of five thousand houses is maintained the annual subsidy will increase by this amount every year. It will be seen therefore that this poses a very large financial problem to Government and this problem can only be solved by continued prosperity and the operation of a quinquenial upward review of rents which would in course of time become fully economic.

CONCLUSION

I have endeavoured in this short paper to give a brief picture of housing conditions in Singapore for the lower income groups and to outline some of the problems.

Perhaps the main problem in the foreseeable future will be one of economics. The technical and structural problems have by and large been solved over a period of twenty years' research and experiment and the most economical use of land and materials has been pretty well established. So also has a balance been achieved between initial cost and maintenance costs. Social problems are such that succeeding generations must each learn by experience to solve as they come along. At the present time there is a world recession and there is also a continual state of tension on the use of nuclear energy. Until these things are stabilised there can never be real prosperity in the world.

The present recession has adversely affected Singapore trade and particularly the basic Malaya industries of tin and rubber. Increasing unemployment in a rapidly rising population is bound to reduce living standards in the next few years, and the ability to pay economic rents, which is low at present, will be lower still. It will be difficult to find the capital to maintain the present housing output and still more difficult to subsidise such housing. The result may be that standards of accommodation may be reduced not by design but by economic necessity. Houses designed for a family of seven may well be overcrowded by joint families of twelve or more as happened to S.I.T. housing during the Japanese occupation. This is perhaps a pessimistic outlook but these things can happen!

As is happening now efforts will be made for budget purposes to reduce minimum standards of construction and occupation. To succumb to these pressures would be the greatest folly. There are certain standards which we must maintain, and which if maintained will withstand temporary vicissitudes. In the long run the people will demand these standards of better housing and to achieve them will require the pooling of the world's resources of knowledge, skill and wealth.

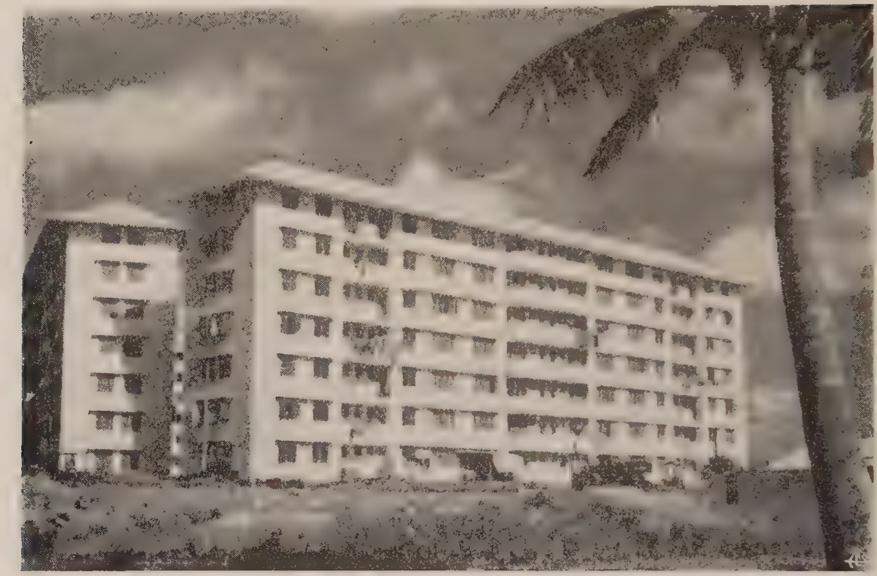

Fig. 1 — 7 storey flats at Red Hill Estate

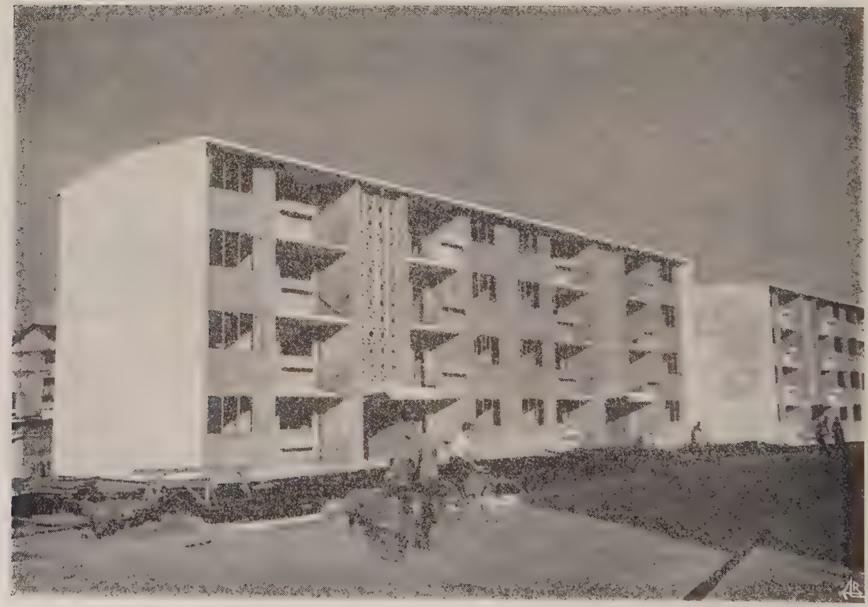

Fig. 2 — 4 storey flats at Princess Estate, Queenstown

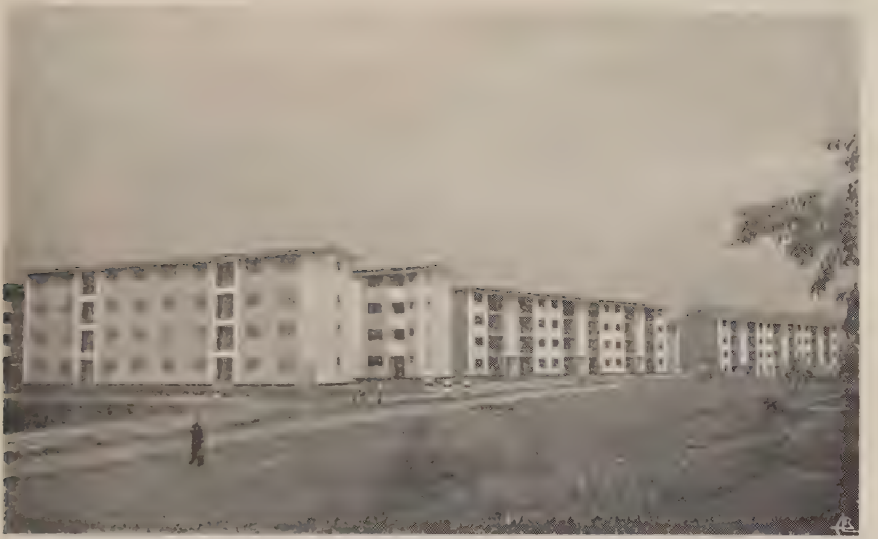

Fig. 3 — Storey flats at Princess Estate, Queenstown

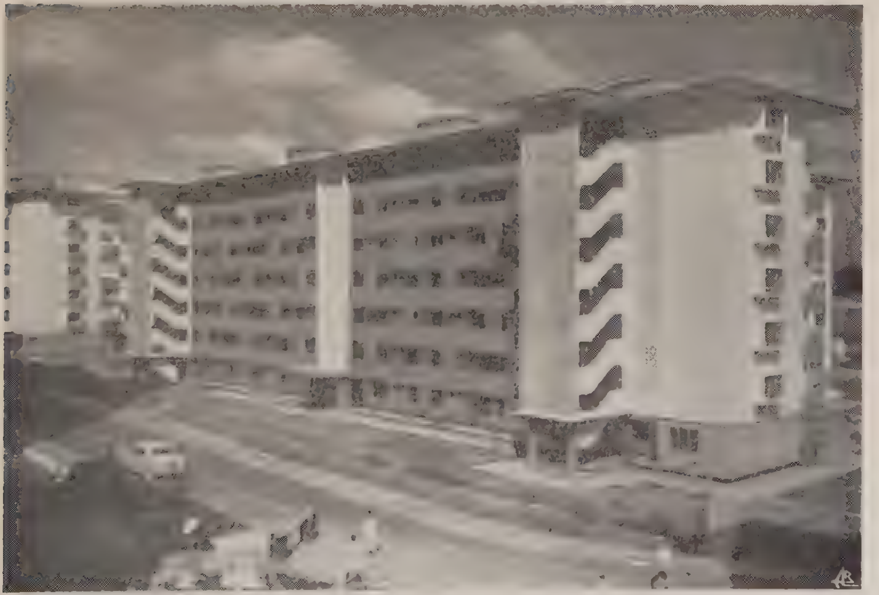

Fig. 4 — 7 storey flats at Princess Estate, Queenstown

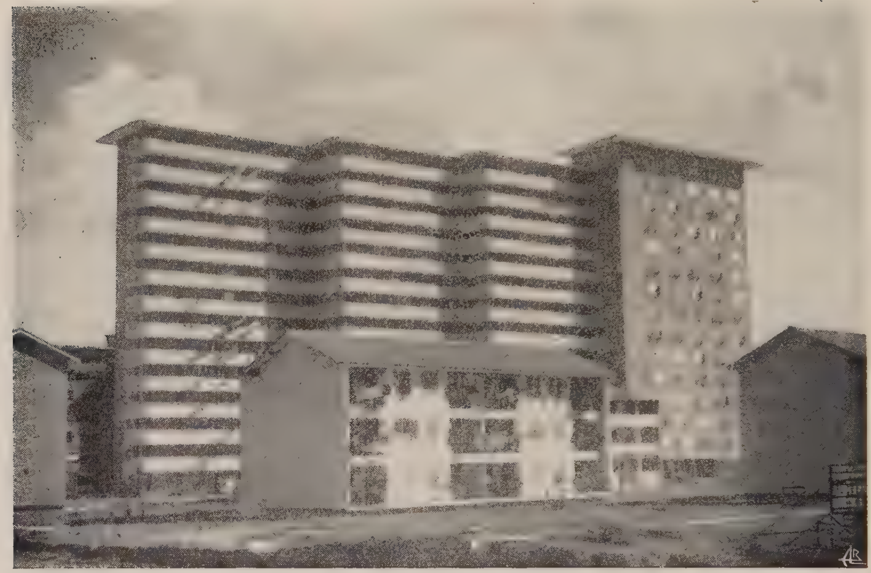

Fig. 5 — "Forfar House" — 14 storey shops & flats at Princess Estate, Queenstown

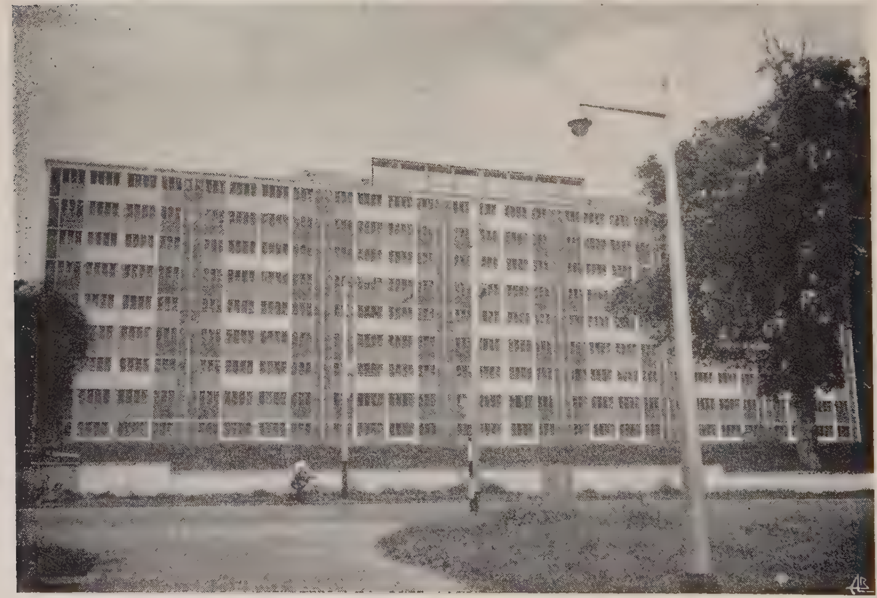

Fig. 6 — 9 storey flats at Windsted Court Estate

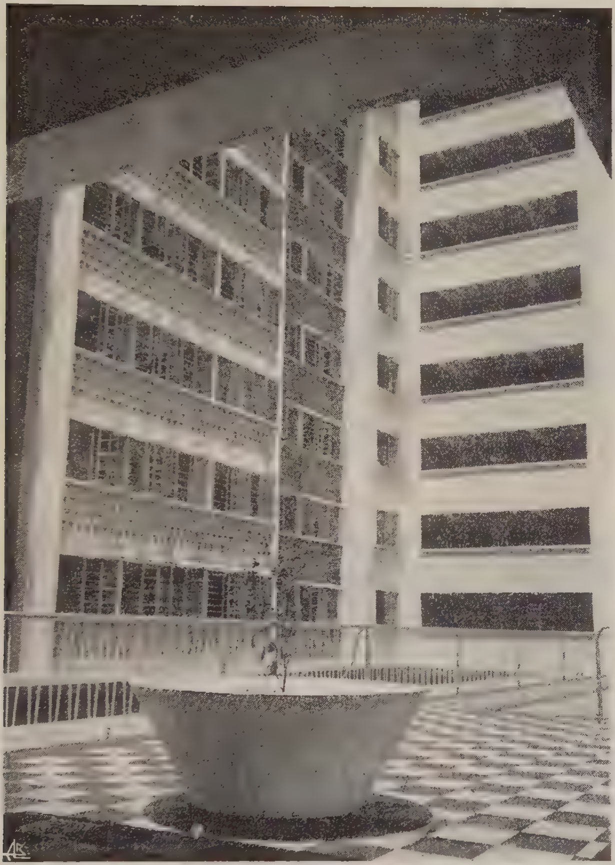

Fig. 7 — "Albert House" — 9 storey shops & flats at Albert Street, Singapore

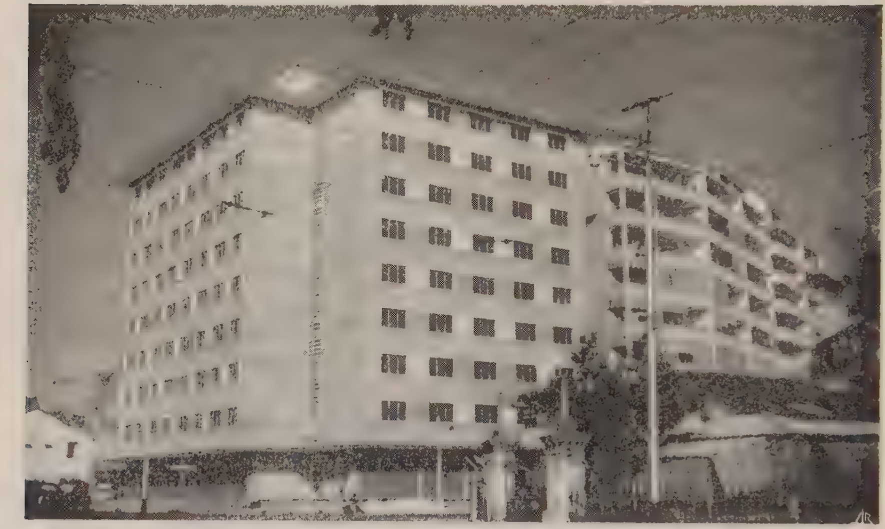

Fig. 8 — 9 storey shops & flats at South Bridge Road

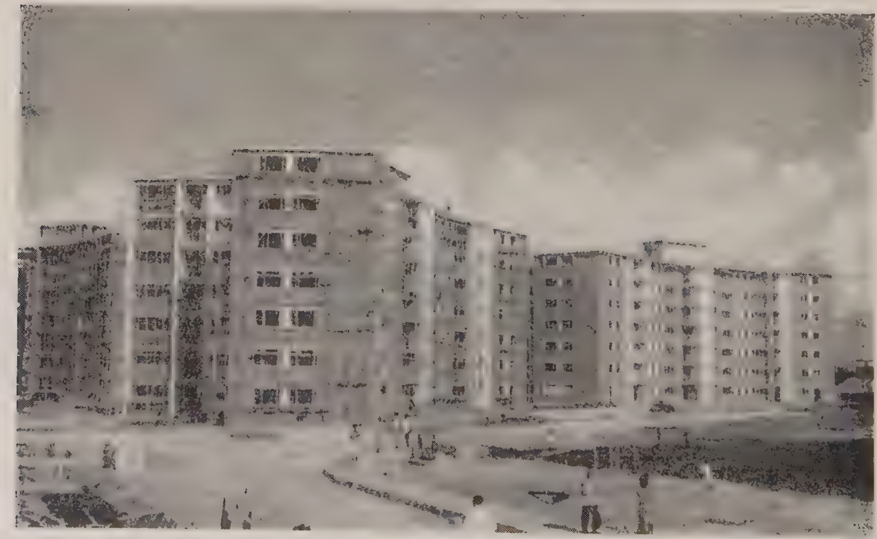

Fig. 9 — 7 storey flats at Kallang Airport

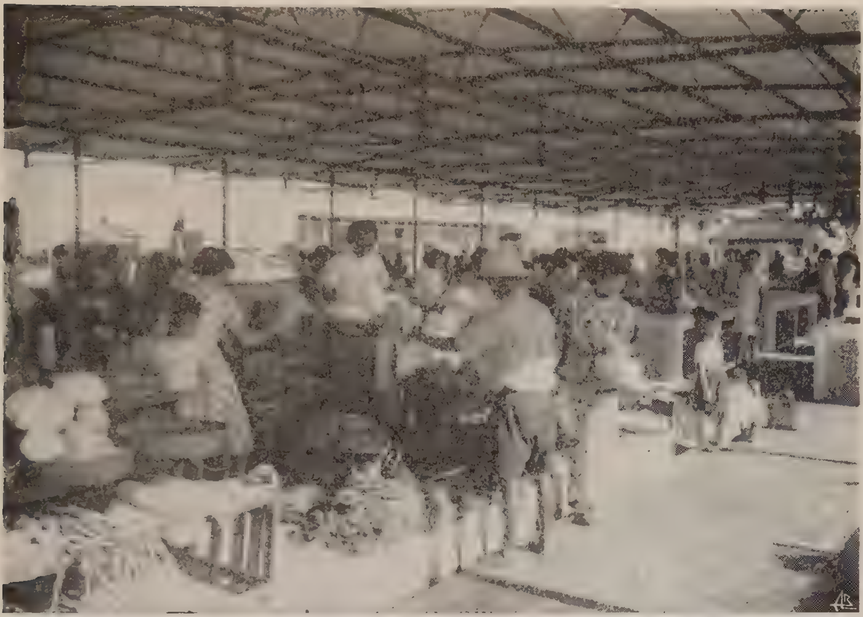

Fig. 10 — Interior of market, Princess Estate, Queenstown

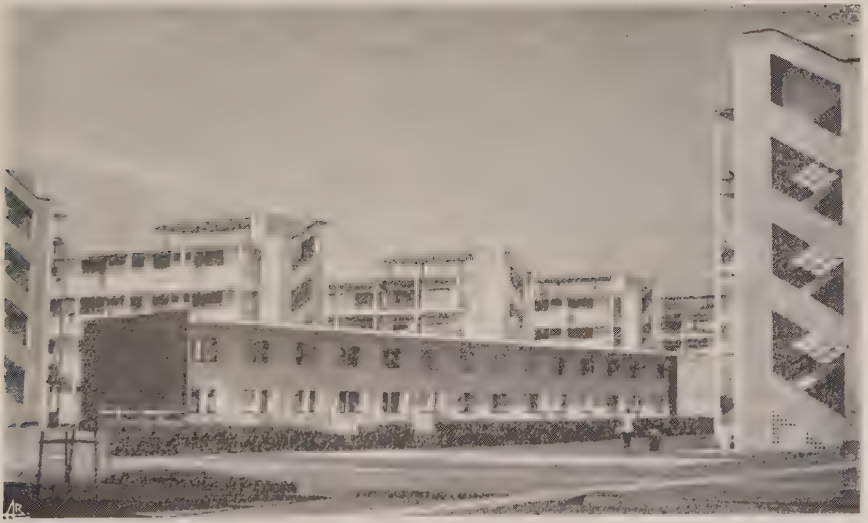

Fig. 11 — 2 storey & 4 storey flats at Jalan Besar Estate

RECENT ADVANCES IN LOW COST HOUSING IN TROPICAL AREAS[1]

G. A. ATKINSON

On Christmas night 1953, over 50,000 pesons lost their homes in the most extensive fire in Hong Kong's history. Nearly all were refugees who had come in large numbers from mainland China since 1947. They were living in overcrowded huts built of timber, cardboard, flattened metal sheet and other waste materials. Sited haphazardly on hillsides and in ravines too steep for normal building, these shacks were packed close together with little air or light, no sanitation and seldom organised refuse disposal. Water had to be carried long distances from overworked common standpipes or, when the season allowed, hillside streams. Pigs, hens and ducks were farmed in and around the shacks which also housed most varieties of cottage industry. Access by ambulance and fire engine was impossible, as was inspection by health and other public officials. Conditions in this and other squatter areas (Fig. 1) and the measures taken by the Government's Resettlement Department to house nearly 250,000 former squatters, are described in official reports (1).

Shortage of land and extrem overcrowding make Hong Kong's squatter problem, and the measures taken to solve it, exceptional; but similar difficulties face most other Asian countries, particularly India, Indonesia, Pakistan and Singapore (2). Disorderly, insanitary shack communities in and around towns are not unique to Asia. They are to be met with in many parts of Africa, the Caribbean and Latin America; sometimes also in Europe. In Britain, even, we have the problem of permanent caravan dwellers living in not so very dissimilar conditions.

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Estimates of the number of tropical families living in shack settlements, or in equally unsatisfactory overcrowded tenements, are little more than of academic interest. Information, based on survey, of the ways in which families are housed in tropical towns is scarce.

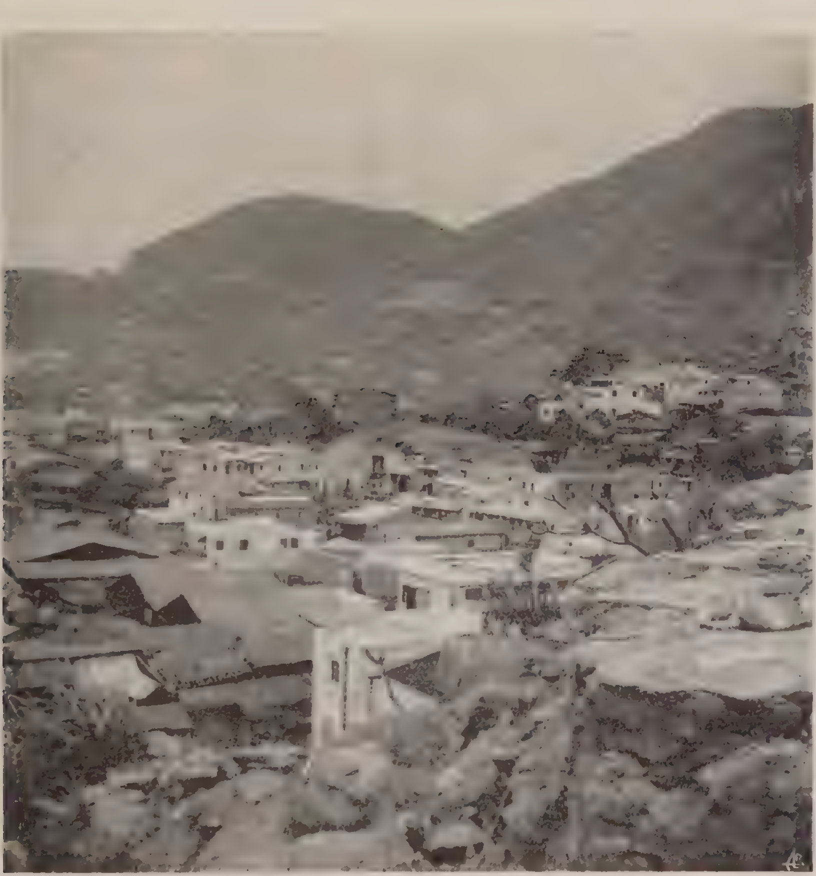

Fig. 1 — Squatter area, Hong Kong (1952): Substantial, though haphazardly sited brick buildings surrounded by timber shacks

Two recent surveys give a picture of conditions in Asian and African towns: Singapore (1953-54) (3), and Jinja, Uganda (1957) (4). In Singapore, one in five of lower-income households within city limits were living in 'attap' dwellings (timber huts with thatch roofs, mostly sited chaotically); one in three in 'shop houses' (uper floors of brick or concrets buildings, usually divided into overcrowded cubicles); and only one in eight in government built and managed housing (Fig. 2). In Jinja, a newer community with a large temporary worker population, one in three lived in African-built and owned housing on the fringe of the town, mostly mud huts with thatch or metal roof not conforming to building rules and sited haphazardly. One in three lived in housing provided, usually free of rent, by employers and varying greatly in quality from neat police quarters to old, neglected labour lines. Between one in four and five lived some distance from the town in rural surroundings. Only one in ten was showed in estates built and run by government as a social service.

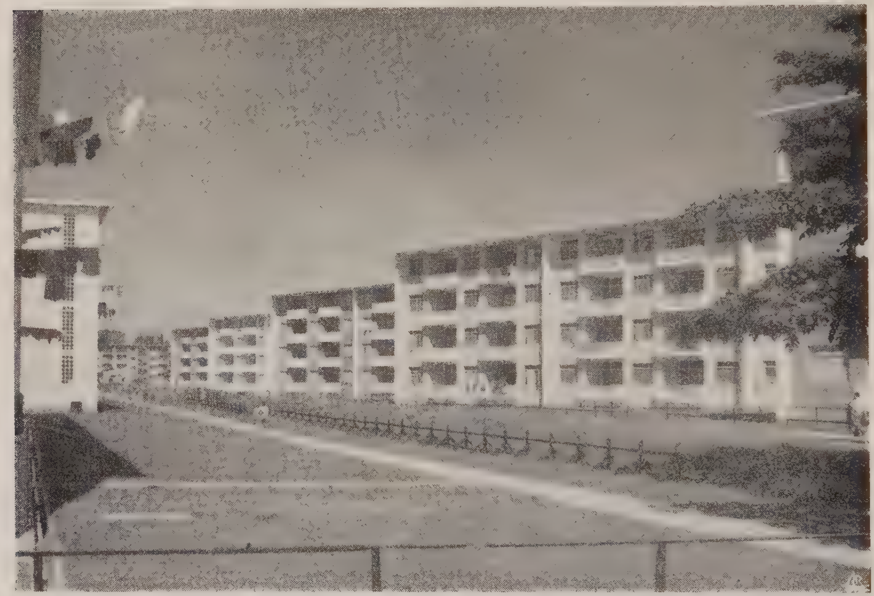

Fig. 2 — Social Housing, Singapore (1953): Four-storey flats, Tiong Bahru Road, Singapore, built and managed by the Singapore Improvement Trust (Net floor area of each three-room flat (including verandah) 730 sq. ft.)

These figures, from places where social housing activity is above average (5, 6), indicate the scale of the problem facing tropical countries. Few governments can afford money or technical resources to house as a social service more than a minority of urban families. Only model or large employers are willing or able to house satisfactorily many of their workers. The mass of town dwellers must, therefore, depend on their own initiative or on that of private entrepreneurs for shelter, often to be found in shack settlements or overcrowded cubicles. Where public authority is able, it condemns and clears such areas. But poverty — private and civic — often precludes rehousing all displaced in permanent buildings at an acceptable standard. Those not so housed take any compensation offered for disturbance and move to spots where authority is less able or willing to control them.

Deficiencies in these procedures, especially under pressure of disaster or the urgent need of squatter occupied land for essential development, have prompted a reappraisal of methods of clearance and compensation (7, 8). These and complementary measures to control new, and to administer and improve existing settlements are discussed below. Broadly speaking, they have meant accepting the realities of the situation, particularly the fact that many town dwellers will have to live in huts for some years to come and that, consequently, this form of mass housing cannot be ignored or condemned outright.

Building rules in most tropical countries are modelled on western examples. Enacted first at the beginning of the century when, at least in many African and Asian towns, protection of European officials and traders against tropical disease dominated urban sanitary policy, they set standards for domestic building related more to what was desirable in an official quarter than essential for mass housing (9) (for example, former Hong Kong rules permitted lower ceilings in servants' than main rooms). The rules, especially when brought up to date in light of recent knowledge, have largely prevented shoddy building and, especially in crowded central areas, given protection against risk to life of structural collapse and fire. They have also ensured reasonable sanitary conditions. But, for many families, dwellings built to legal standards are too costly to occupy except when seriously overcrowded.

Many, therefore, have to live in huts built illegally or beyond town limits. Though more flimsily built and haphazardly sited, these dwellings are not without merit. They are likely to be less overcrowded than more solid, 'legal' buildings. (In Singapore, for example, 81 per cent of the 'shop houses' in the sample surveyed were overcrowded compared with only 59 per cent of the 'attap' huts (3). At Jinja, greatest overcrowding was found in the usually well built employer housing) (4). Southall and Gutkind (10) found in Kampala that many Africans prefer this kind of housing to the more sanitary and orderly government estates.

To lessen the contrast between solidly built — but probably overcrowded — urban dwelling and their flimsier illegal fellows, some tropical governments have adopted graded building rules which permit less substantial houses in selected parts of towns and facilitate bringing fringe settlements under control. For example, following discussion by the East Africa Royal Commission Uganda, in 1955, introduced a procedure by which areas are set aside where modified building rules apply. Three grades of rules have been adopted: Grade I, the normal standard of urban building; Grade II, permitting small houses (no larger than 1000 sq. ft. — 92.9 m^2 — in floor area) with mud walls but a durable, non-inflammable roof; and Grade III, permitting temporarily the erection of still simpler houses with thatch roofs. In the modified rules, siting and the provision of kitchen, wash space and latrine are considered more important than materials and methods of construction (Fig. 3). Grade II rules permit habitable rooms as small as 80 sq. ft. or 7.4 m^2; no minimum size is specified in the Grade III rules (11).

The ministerial circulars which brought the new rules to the notice of urban authorities in Uganda required that the selection of areas where the simpler rules would apply should be made in consultation with the government town planner, who would prepare layout plans, and the Director of African Housing, who would either undertake direct control of the scheme — in some principal towns — or assist with advice, model house plans and, on occasion, finance for initial service. The health authority had also to be consulted. Arrangements were made for granting leases to householders — for ten years or more in Grade II areas. Progress in greater Kampala has been described by Russel and Hather (12); in Kampala and Entebbe by Hather and Oram (13).

Similar procedures have been adopted or recommended in other countries (14). In February 1958, at a conference on urban problems in East and Central Africa hold at Ndola, Northern Rhodesia, four out of six of the territories represented were reported to have introduced grade building rules, some very recently. Five of the six were reported to have arrangements for helping householders to build to simplified or normal rules.

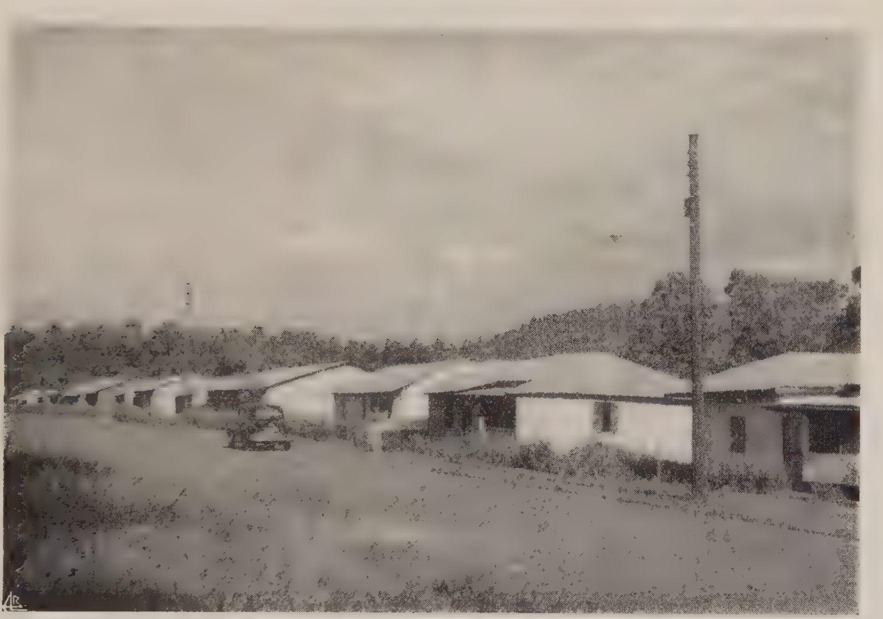

Fig. 3 — Ntinda Estate, Kampala, Uganda (1957): African-built houses to Grade II Building Rules

The fixing of standards for mass housing in tropical countries was studied at length during the 1954 United Nation Regional Seminar on Housing and Community Improvement in Asia and the Far East (15). The guiding principles recommended merit quotation at length and are appended to this paper. Attention is drawn to the need to provide each dwelling with its own latrine and wash space; to define living spaces by a suitable floor area, based on the size of families mostly to be housed, rather than by fixed room sizes; and to make best use of outdoor space.

Often, when an organised system of sanitation has been provided for mass housing, communal latrines are built. Even when well maintained — and good maintenance is costly, families dislike them. Neglected, they are a source of illhealth. The experience of the Uganda African Housing Department (6), which has been replacing modern communal latrines with individual pit latrines on its social housing estates, confirms the author's personel observation that most tropical families keep clean individual latrines — however primitive — but communal sanitation is rarely satisfactory.

At low densities (up to 50 persons/acre or 120/hectare), deep pit or borehole latrines give satisfaction under normal sub-soil conditions. At higher densities (say up to 150/acre or 375/hectare), they are possibly still to be preferred for mass housing where the sub-soil is suitable — and a factor in site selection should be suitability for pit sanitation, where piped water is available, but waterborne disposal too costly. At these densities, especially if water is cheap and in ample supply, separate disposal may be needed for waste water. At still higher densities, or where unfavourable sub-soil conditions occur like water-logged ground or rock, there may well be no alternative to fully water-borne sanitation. Intermediate systeme like the aqua-privy have not always proved satisfactory (16). The selection of a suitable system of sanitation depends on local circumstances, social and economic as well as technical. Research workers in tropical hygiene could assist by providing basic information.

A comparative scale of living space commonly available under various circumstances in different parts of the world is suggested in Table 1. It shows a range (in m^2 of gross floor area/person) from over 18 for middle-income professional families, 13-17 for postwar social housing in Western Europe, 8-12 for recent housing in Eastern Europe and some Mediterranean countries, 4-9 commonly provided in mass housing in Africa and the Caribbean, 3-5 for Hong Kong and other densely crowded Asian cities, to 0.75-2.75 for seriously overcrowded conditions.

In an earlier review (17), the author suggested for low-cost tropical housing that, while minimum areas should be based on the space per person required for sleeping, the number of rooms available to a household was important if there was to be a measure of privacy and amenity. In a warm climate, many household activities take place outdoors, especially where there is a private yard. The dwelling, at least with the less well off, serves mainly for sleeping and storage;

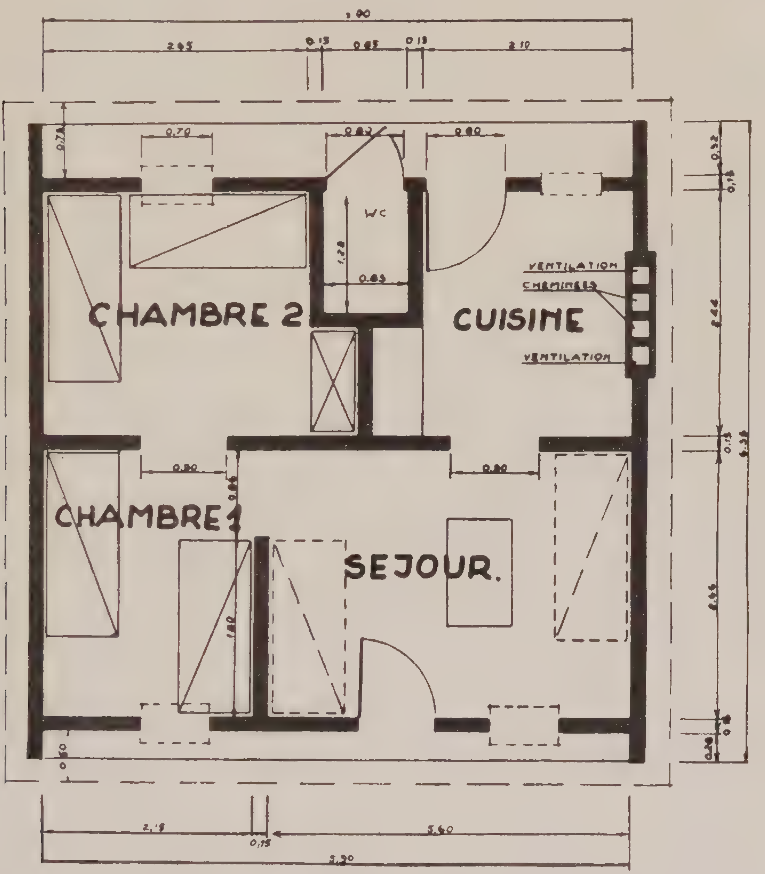
CHAMBRE 2
WC
CUISINE
VENTILATION
CHEMINEES
VENTILATION
CHAMBRE 1
SEJOUR.
0,70
0,80
1,28
0,85
0,80
0,80
1,80
0,15
5,60
5,90

Fig. 4 — Terrace House, O. C. A., Belgian Congo (1956): Plan of single-storey terrace house, Office des Cités Africaines, Belgian Congo (Type T15, floor area 30.5 sq. m.)

indeed in drier climates it is possible — even preferable in the hot season — to sleep outdoors. Where there is less than 8-9 m² (about 85-95 sq. ft.) per person available to, say, a five person household, it is likely that all the main rooms will be occupied at night, even if one is furnished as a dayroom. Where there is less than 4 m² (about

45 sq. ft.), it becomes difficult to provide two rooms and separate kitchen and bath rooms. At 2 m^2 (just over 20 sq. ft.), there is hardly space at all for cooking or washing, even if beds are in tiers.

In many tropical countries, the majority of less well off households not living under conditions of severe overcrowding are probably now occupying between 4 and 9 m^2 (43-97 sq. ft.) of space per person (Fig. 4). In places where housing is scarce, families given accommodation much in excess of the local average are likely to sub-let part of it. It is suggested that greater attention be paid to facilities like individual latrines and private yards than to attempts to build larger dwellings.

Emphasis on private outdoor as well as indoor space suggests an approach to residential densities aimed at ensuring sufficient space around as well as within dwellings. Stevens (18), in a study of densities in housing areas, relates these to total living space (outdoor and indoor) and space per person within a dwelling. Many factors influence choice of a total living space standard avalability of land; space for access, light and air, household activities, recreation and, sometimes, food-growing; prevention of spread of fire; safe disposal of soil water and other waste matter; etc. In turn, such factors may depend on local social and economic conditions, climate and soil.

The total living space available in a number of tropical housing schemes is listed in Table 2. Per person, it ranges from 40 sq. ft. (3.7 m^2) in Hong Kong resettlement tenements (1) to 500 sq. ft. (45 m^2) or more in semi-urban schemes with space for food-growing or even, as in the Singapore scheme (8), small-scale market gardening. Corresponding housing densities range from 2100 person/acre (5250/hectare) to 30-40 (75-100) or less.

High densities like those in Hong Kong and some other Asian cities are only possible with very high occupancy rates and a very low standard of total living space. Hong Kong experience shows that such densities in well-managed, multi-storey concrete buildings with engineered services may be technically adquate for the emergency housing of homeless people; but they hardly provide any privacy or social amenity.

Single-storey houses with plots of less than 1000 sq. ft. (90 m^2) like the Karachi and Indian schemes in Table 2 may, if not seriously overcrowded, give densities of 200-300 person/acre (500-750/hectare).

Built in fire-resistant construction and provided with engineered services, housing at such densities seems technically acceptable; with walled yards it gives reasonable privacy at a total living space per person of 100-200 sq. ft. (9-19 m^2). The two-storey Kenya scheme would provide much the same conditions.

In a number of Africain towns, mud-walled houses with metal, or sometimes thatch, roofs are built ond 1500-2000 sq. ft. (140-185 m^2 plots (Fig. 5); typical is the example from Zanzibar. When it is occupied by one family (5-10 persons), densities may range from 100-200/acre (250-500/hectare) and total living space per person from 200-400 sq. ft. (18.5-37 m^2). Where soil conditions are favourable — e. g. the fissured coral of Zanzibar and Mombasa — and water is piped, owner-buil' housing with pit sanitation seems to have no noticeable shortcomings; but, especially where rainfall is heavy or water consumption high, storm and, possibly, waste water drainage may be needed. However, similar buildings may be used as lodging houses accommodating many people. Densities rise to 300 or more/acre (750/hectare). Then sanitary conditions deteriorate.

Building rules do not usually distinguish between one-family and lodging houses, though the distinction may effect densities and, consequently, health. As Table 2 shows, the Uganda Grade II Rules, and the recommended standards for Tanganyika, permit houses up to 1000 sq. ft. (93 m^2). While it is commendable to encourage such relatively large dwellings if occupied by one family, a many-roomed lodging house may be built accommodating from 20-25 persons (at usual occupancy rates), or up to 50 persons when overcrowding is serious. High densities, low standards of total living space (less than 100 sq. ft. — 9 m^2), and unhealthy conditions result. In drafting building rules for mass-housing, it is advisable to keep maximum permissible floor areas down to a figure — say 500-650 sq. ft. (45-60 m^2) under present conditions in many tropical countries — which favours one-family rather than lodging houses. Plot sizes will correspond. Where lack of land and other factors require fairly high densities, plot sizes may range from 1500-1800 sq. ft. (140-170 m^2) giving about 1/3 coverage or less. Where there is space and a tradition of food-growing, plots may be double — say, 3000-4000 sq. ft. (275-350 m^2); they can then be divided satisfactorily when the community grows and densities have to be increased. Separate rules,

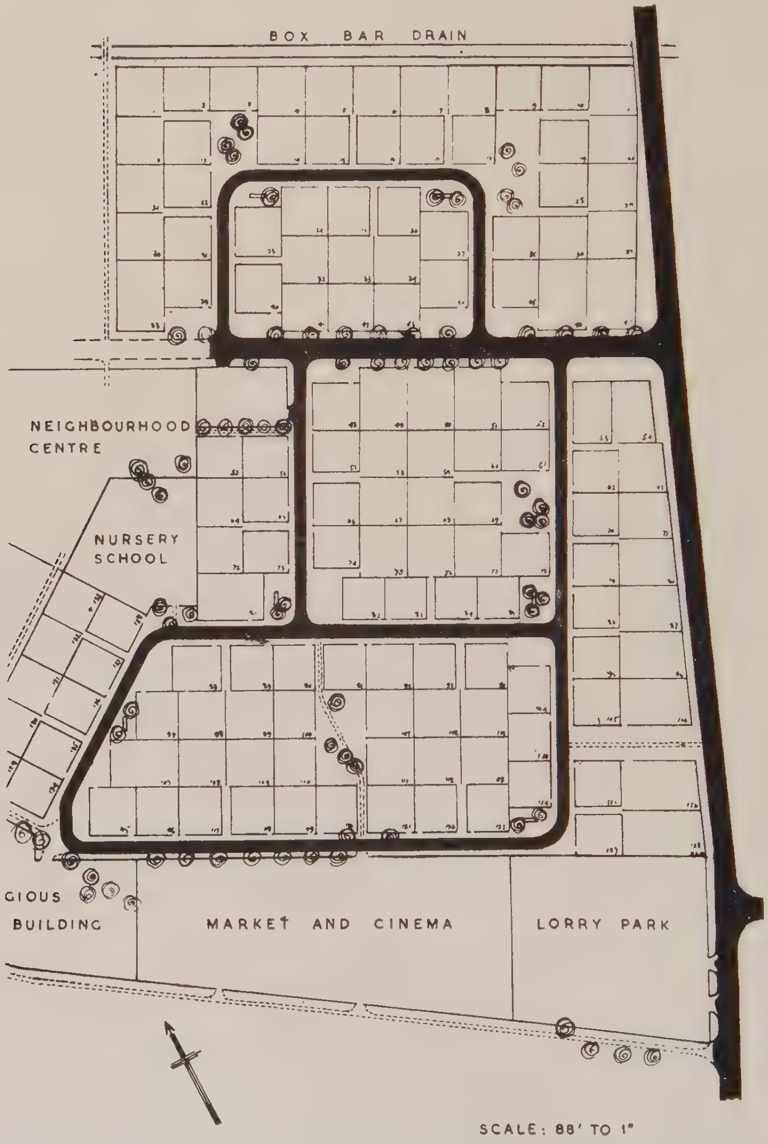
BOX BAR DRAIN
NEIGHBOURHOOD CENTRE
NURSERY SCHOOL
GIOUS BUILDING
MARKET AND CINEMA
LORRY PARK
SCALE: 88' TO 1"

Fig. 5 — Housing Layout, Bathurst, Gambia (1955): Suggested Layout of the Crab Island reclaimed area for low cost housing. (Plot sizes, 2,750 and 3,025 sq. ft.)

with appropriate safeguards, are likely to be required for lodging-houses.

The application of aided self-help methods to the housing of tropical peoples was discussed at length in the United Nations Housing Bulletin No. 6, which also contains details of schemes in many countries and a check list of items to be considered in their organisation (19). Asian and Latin American experience was reviewed in later Bulletins (No. 9 and No. 10) (20, 21). Ways of encouraging house-ownership, and particularly home-building, in British African territories and also in Ghana, where a roof loan scheme is in operation, have been described (22, 23). Much has been written on the subject in the United States (24). Methods of assistance include technical advice, help with materials, tools and skilled labour, the lease of a building site developed with appropriate services, and the organisation of cooperative groups of self-help builders. The choice of a particular method, or combination of methods, will depend on local circumstances — eco-

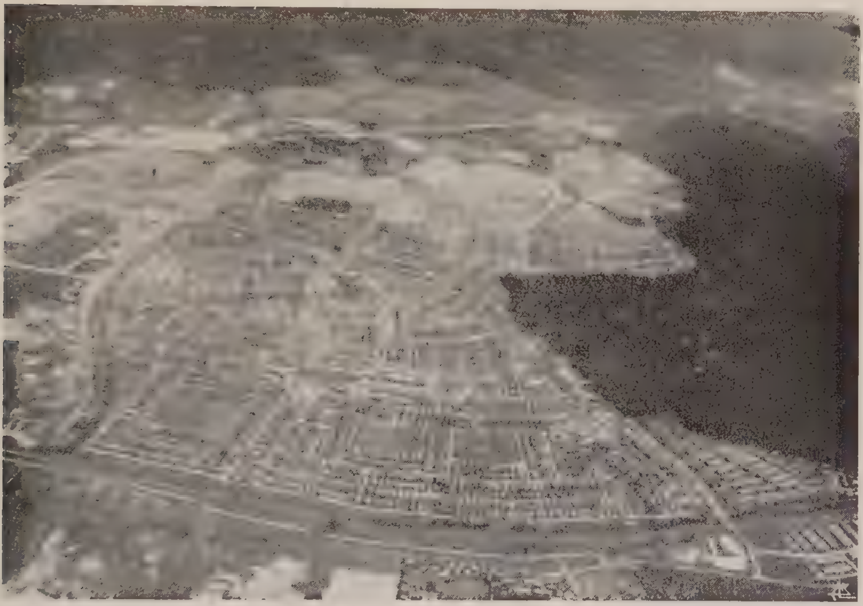

Fig. 6 — Petaling Jaya, Kuala Lumpur, Malaya (1957): Air view of new satellite town. (Photograph by Federal Town Planner)

nomic, social and technical. Specially it will depend on the wealth, or poverty, of the community concerned.

Even at higher densities (over 100 persons/acre), mass housing schemes of the occupier-built type require relatively large areas of land. It may be possible to plan these settlements as satellite communities as, for example, the new villages near many Malayan towns and Petaling Jaya, the more substantially built satellite of Kuala Lumpur (25) (Figs. 6 and 7). But topography and other factors may prevent this. Then it may be necessary to house even the poorest families at high, or very high densities. Reference has already been made to the Resettlement Department's tenements at Hong Kong (1) (Figs. 8 and 9). Exceptional conditions have necessitated very low space standards (planned floor areas as low as 24 sq. ft./person, with total living areas in and around the tenements of only 36 sq. ft./person or, if the roof area is included, 40 sq. ft.). Efficiently built in cross-wall concrete construction, these seven-storey 'walls-up' tene-

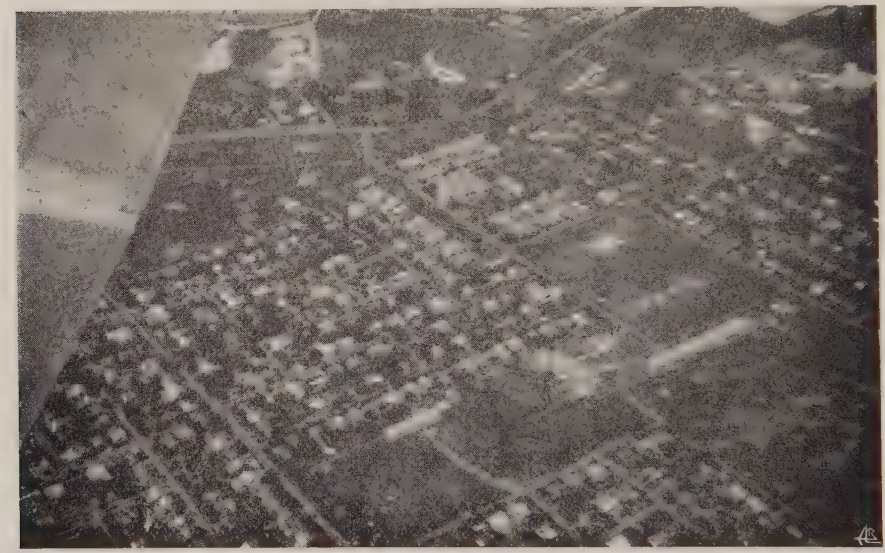

Fig. 7 — New Village, Near Kuala Lumpur, Malaya (1956): Built to house Chinese squatters, now a permanent settlement. Building standards resemble those of the Uganda Grade II Building Rules

ments are costing just over £ 110 (US $ 310) a room. Allowing for management and maintenance costs, ground rent, and capital repayment over 40 years, the rooms are being rented at 17s. 6d. (US $ 250) a month, a figure within the means of most of the poorest households. General health in the territory appears to be improving despite the low housing standards (26). That these need to be revised as soon as the urgent problem of housing the homeless is solved is fully appreciated. Plans are ready, and prototype flats have been built, for the conversion of single room tenements into self-contained flats at roughly double present space standards (Fig. 10). At the same time, the Hong Kong Housing Authority (27) and other organisation have been building large numbers of flats for workers with higher incomes (Fig. 11).

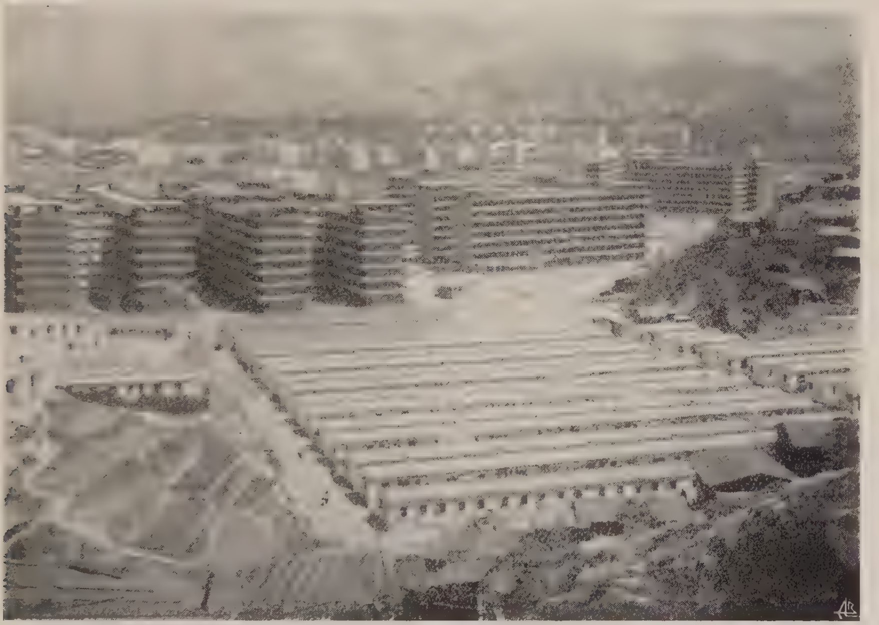

Fig. 8 — Resettlement Housing Hong Kong (1956): Single-storey terrace houses and seven-storey tenements built for the Hong Kong Resettlement Department (to house refugees)

Space does not permit discussion of technical advances in house building. In a review of building techniques overseas, the author suggested that modernisation of local materials industries, and particularly establishment of cement and associated concrete products industries, were likely to be more advantageous to low-cost housing than, for example, the import of factory-made buildings or the introduction of novel construction methods (28). Experience seems to confirm this (29). Greater efficiency in building is likely to be secured by improvements in training at all levels and by planning operations to make greatest use of the varying skills of workers (30, 31).

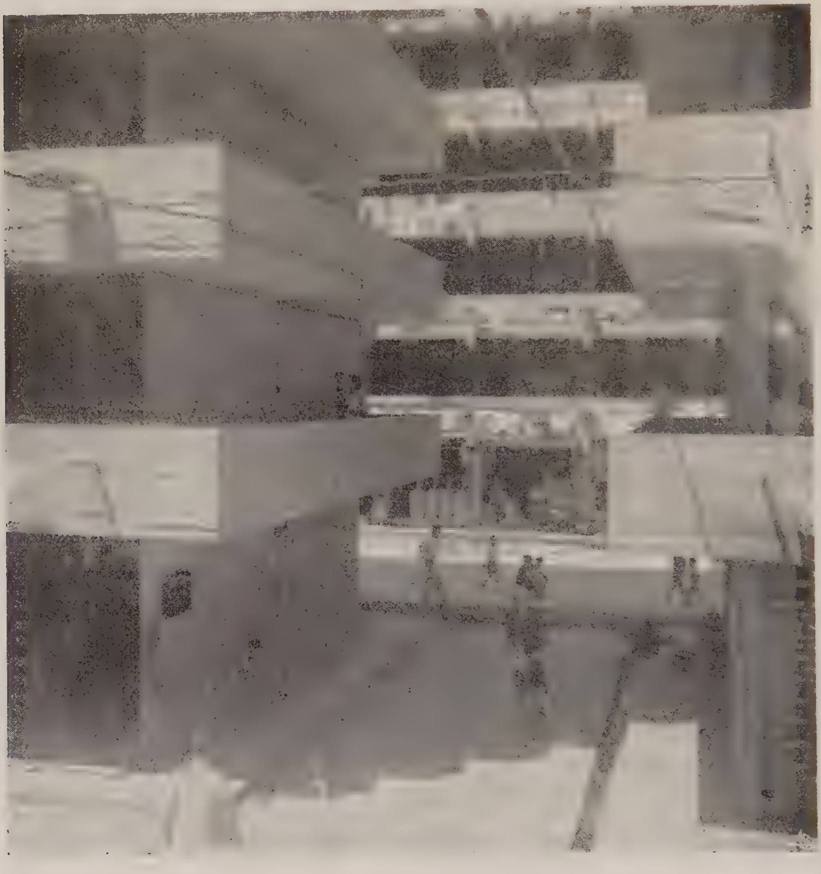

Fig. 9 — Resettlement Tenements, Hong Kong (1956): Close-up view of seven-storey blocks housing families at densities of over 2,000 persons/acre

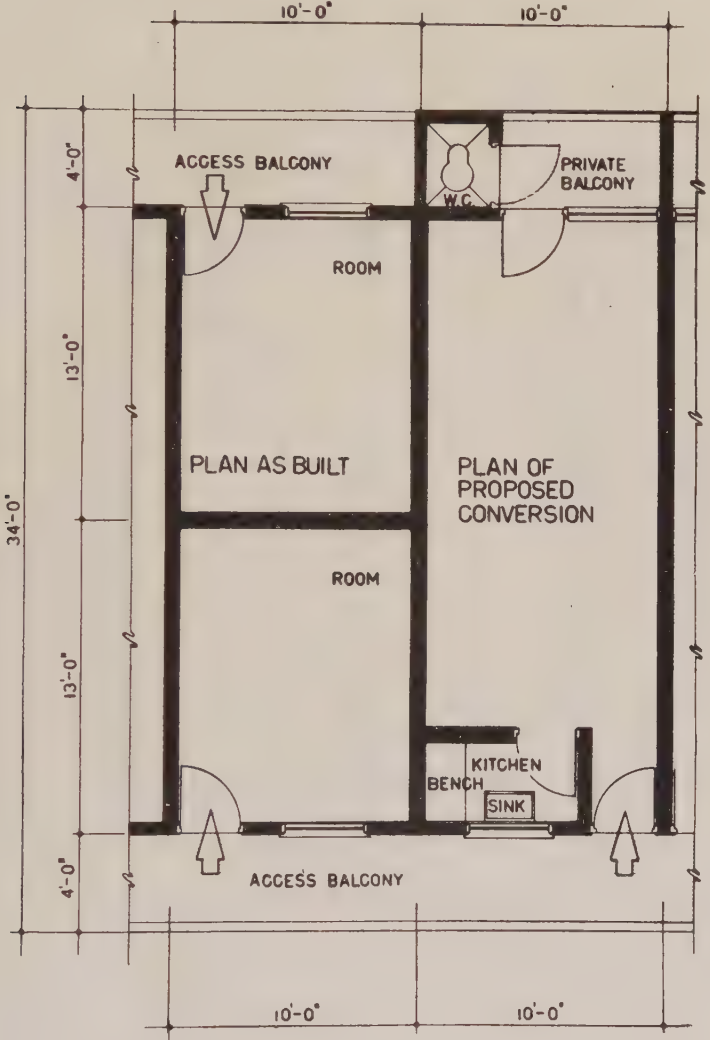
10'-0"
10'-0"
4'-0"
13'-0"
13'-0"
4'-0"
34'-0"
ACCESS BALCONY
W.C.
PRIVATE BALCONY
ROOM
PLAN AS BUILT
PLAN OF PROPOSED CONVERSION
ROOM
KITCHEN
BENCH
SINK
ACCESS BALCONY
10'-0"
10'-0"

Fig. 10 — Plan, resettlement tenement, Hong Kong: Space standards are at present as low as 24 sq. ft./person; but plans provide for improvement in the future

Technical aspects of tropical building are receiving increasing attention by building research and similar institutions (32), a number of which are listed in Appendix 2.

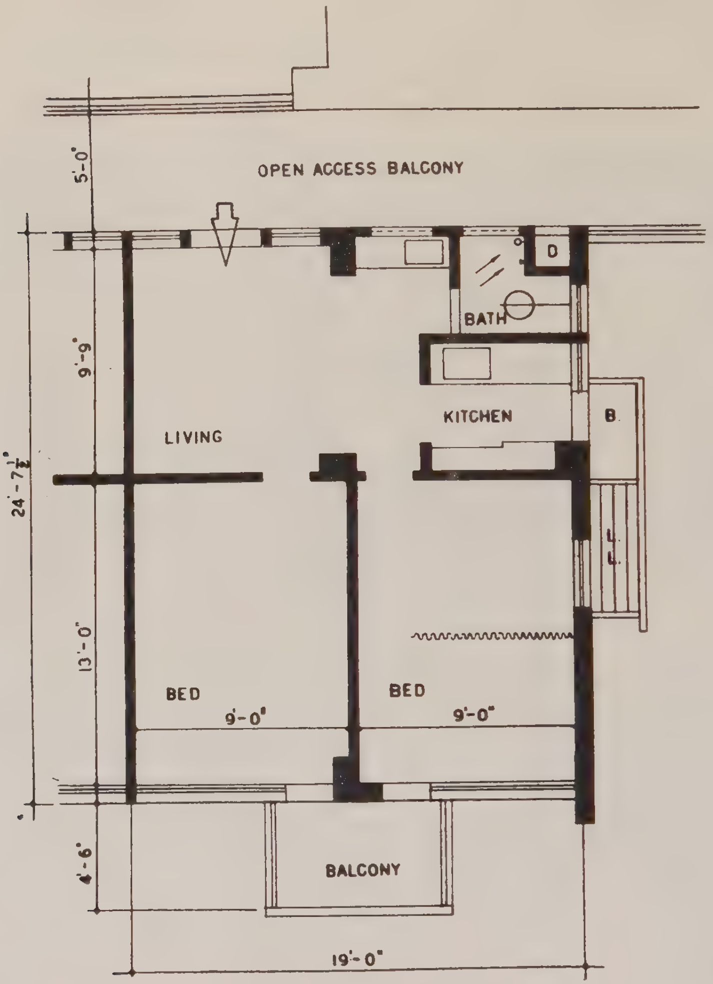
OPEN ACCESS BALCONY
5'-0"
9'-9"
24'-7½"
13'-0"
4'-6"
LIVING
BATH
D
KITCHEN
B.
BED
9'-0"
BED
9'-0"
BALCONY
19'-0"

Fig. 11 — Plan, North Point Flats, Hong Kong (1957): Typical flat being built for the Hong Kong Housing Authority (floor area 413 sq. ft.)

Acknowledgments — The work is part of the programme of the Building Research Board, and acknowledgments are made to the Director of Building Research for permission to publish the paper.

TABLE 1 — *Comparative scale of dwelling space commonly available in different parts of the world*

(The following tentative scale is based, wherever possible, on that usually provided, or commonly available, for a household of 5 under various circumstances in different countries. Figures are usually based on gross floor areas within outside walls on all floors occupied by th household. Overall building areas will normaly be 10-15 per cent larger)

Type of household and dwelling	Gross floor area, 5-person dwelling		
	Total area	Area per person	
		m²	sq. ft.
Families of professional groups in W. Europe	—	*over 18*	*over 200*
Social housing in post-war W. Europe	—	*13–17*	*140–180*
English Housing Manual (1944)[I]:			
a) houses	800–900 sq. ft.	—	160–180
b) flats	740–790 sq. ft.	—	150–160
U. K. Ministry of Works (1944)[II], prefabricated bungalow	623–643 sq. ft.	—	156–160 *
Netherlands (1952-54)[III], minimum flats at Vlaardingen	63 m²	12.6	—
Social housing in pre-war W. Europe	—	*11–14*	*120–150*
English Ministry of Health (1936)[IV], minimum cottage	760 sq. ft.	—	152
London C. C. (1935-37)[V], terrace cottage	722 sq. ft.	—	144
London C. C. (1934)[V], flat in redevelopment scheme	658–690 sq. ft.	—	132–138 110–115 **
Netherlands (1930-34)[III], workers, housing	—	12–12.5	—
Housing in E. Europe und parts of the Mediterranean area	—	*8–12*	*90–110*
Latin America (1953)[VI], recommended minimum for social housing	60 m²	12 10 **	—
Greece (1951)[VII], nucleous house	47.2 m²	11.8 * 9.6	—
France (1928)[IV], Ministry of Labour, minimumflat	45 m²	11.2 * 9	—

Type of household and dwelling	Gross floor area, 5-person dwelling		
	Total area	Area per person	
		m²	sq. ft.
Warsaw (1953)[viii], newly built flats	35.3–44.8 m²	8.8–11.2 **	—
Jugoslavia (1949–50)[ix], average dwelling space:			
b) urban	—	11.6	—
a) rural	—	7.9	—
W. Germany (1948)[x], emergency post-war housing	40 m²	10 * 8	—
Hungary (1954)[ix], average useful floor space,*** Greater Budapest	—	8.6	—
Soviet Union (1954)[ix], average useful floor*** space, urban areas	—	7.2	—
Housing in Africa, the Caribbean and similar tropical areas	—	*4–9*	*45–100*
S. Africa (1951)[xi], Nationl Housing Commisson, Type NE 51/9	596 sq. ft.	—	100 **
Malaya Housing Trust (1956)[xii], Type A 3/2	495 sq. ft.	—	99 100 *
Barbados Housing Authority (1957)[xiii], Type 100	400 sq. ft.	—	80
Belgium Congo (1956)[xiv], O. C. A, Type T 15	30.5 m²	7.6 * 6.1	—
Kenya (1956)[xv], Makongeni, Nairobi, 5 bed house	350 sq. ft. including varandah	—	70
India (1954)[xvi], Health Development Committee, recommended minimum	—	—	60
Jamaica (1943)[xvii], Census Repor (average dwelling size / average household)	—	—	55
Tanganyika (1957)[xviii], Township Sanitation Report, recommended minimum	—	—	50
West Indies (1944)[xix]. Recommended for new low-cost housing	—	—	48
West Indies (1944)[xix]. Recommended overcrowding standard (also in other tropical countries)	—	—	40

Type of household and dwelling	Gross floor area, 5-person dwelling		
	Total area	Area per person	
		m²	sq. ft.
Housing in some large Asian cities	—	*3–5*	*30–55*
Hong Kong (1955)[xx], legal minimum	—	—	35
(Static caravan: U.K.)	(128–192)	—	(32–48 *)
(Touring caravan: U.K.)	(96–112)	—	(24–28 *)
Hong Kong (1954)[xxi], squatter resettlement tenements	120 (excluding gallery and communal sanitary space)	—	24
Conditions of serious overcrowding	—	*0.75–2.75*	*10–30*
Dacca, E. Pakistan (1953–54)[xxii], average space in factory housing	—	1.3–2.0	19.3
(Crowded youth hostet dormitory with 2-tier beds[xxiii])	—	—	—
(European railway «tourist» class sleeping compartment 3-berth[xxiii])	—	(0.76) (1.1 with corridor)	—
Calcutta (1946)[xvi], «bustee» slum occupied by 8–10 persons	80 sq. ft.	—	8–10

* Based on 4-person household.

** Based on 6-person household.

*** Useful floor space: net floor space of living rooms, bedrooms, kitchens, entrance halls, bathrooms and toilets.

TABLE 2 — *Total living space provided in some tropical housing schemes*

(In the following table, plot size is related to gross floor area of dwelling and estimated number of occupants. Where dwelling may serce as a lodging house, this conditions was noted)

Type of scheme	Gross floor area of dwelling (sq. ft.)	Estimated no. of occupants (if overcrowded or as lodging house)	Area of plot (sq. ft.) (plot coverage)	Total living space (sq. ft./person)
Hong Kong: Resettlement tenements in 5–6 storey concrete building with engineered services [xxi] . . .	120 (excluding communal sanitary space)	5–6	—	36 (40 with roof space)
Hong Kong: Housing Authority, 11-storey flats in concrete with engineered services, including lifts [xx]	413 average	8 average	—	58 (63 with roof space)
Karachi: proposed refugee housing in 1-storey court-yard plan, permanent materials and probably engineered services [xxiv].	275	6	720–990 (30–40 %)	120–165
Kenya: 2-storey, permanent materials with engineered services and walled yard [xxv]	960	12	1350 (35 %)	132.5
India: Ministry of Labour, 1-storey terrace or semi-detached brick houses with walled yard and engineered services [xxvi]	250–480	5–6	800–950 (30–40 %)	135–190
West Indies, recommended for 1-storey detached houses, probably timber with pit latrine and standpipe water [xix]	500	5–10	1500 (33 %)	150–300
India: Ministry of Labour, 1-storey semi-detached owner built but with brick kitchen and party walls, pit latrine [xxvi]	500	5–10	1625 (30 %)	162.5–325
Zanzibar: Ngambo, detached African-built 1-storey house, semi-permanent materials with pit latrene and water from standpipe [xxvii]	660	5–50	1900 (35 %)	190–380

Type of scheme	Gross floor area of dwelling (sq. ft.)	Estimated no. of occupants (if overcrowded or as lodging house)	Area of plot (sq. ft.) (plot coverage)	Total living space (sq. ft./person)
Tanganyika, recommended for African built housing, 1-storey, sem-permanent materials with pit latrine, probably water from standpipe [xviii]	1000	5–10 * (20)	2000 (50 %)	200–400 * (100)
Uganda: Grade II building rules, detached African-built 1–storey house, semi-permanent materials with pit latrine and water from standpipe [xxviii]	1000	5–10 * (20)	2800 (35 %)	280–560 * (140)
India: HERL village house, detached with mud walls/thatch roof, pit latrine [xxix]	500	5–6 * (10)	1800 (28 %)	300–360 * (180)
India: Delhi Exhibition, Bengal Engineering College model house, 1-storey semi-detached built with planed open space for food growing and household activities [xxix]	600	5–6 * (10)	2500 (24 %)	320–500 * (250)
Latin America: CINVA rural detached, 1-storey built by family; probably pit sanitation [xxx]. . . .	684	5–6 * (10)	2325 (29 %)	388–465 * (232)
Singapore: semi-urban smallholder resettlement scheme in timber/thatch with pit sanitation [xxxi] . .	530	5–10	4336 (12 %)	434–867
S. Africa: detached house in permanent materials with engineered services, planned garden for food growing and recreation [xi]. . . .	625	6 * (10)	3125 (20 %)	520 * (312)
S. Africa: detached house, planned garden for food growing with pit latrine [xi]	650	6 * (10)	3170 (21 %)	530 * (317)

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APPENDIX 1

GUIDING PRINCIPLES IN DETERMINING HOUSING

(From the Report of the U.N. Housing Seminar, New Delhi, 1954:
U.N. Housing, Building and Planning Bulletin N°. 9, September 1955)

1. *House types to be related to local conditions.* — A housing programme cannot mature successfully unless the souse types adopted (both in regard to accommodation and construction) are related to the economic, social and physical conditions of the country, or district. Happily, in warmer climates it is not technically so difficult to provide shelter. Houses usually do not need to be heated; even in the more humid areas, many activities can take place out of doors. Building can be simpler and, where there is provision for outdoor living, indoor space may reasonably be smaller than the space provided in a temperate or cold climate.

2. *House a home, not just a shelter.* — House types adopted in many countries, particularly when given legal sanction in building regulations and bye-laws, seem largely unrelated to the local climate and way of life and often are unrealistic, having little scientif or social

validity. The question of house types is not solely a matter of providing hygienic conditions; certain minimum social requirements must also be met. They must provide some degree of privacy and amenity — thus making the house a home, not just a shelter. Such house types will, therefore, depend on the way people live, their family organisations, economic status, etc.; they will also depend on the wealth of the country as a whole, on whether the house is in a congested town or in a rural area, and on the climate. Variation in house types between countries, and also within a country, are to be expected. Furthermore, house type are not likely to remain static, but will depend on social and economic development.

3. *Housing codes to establish guiding principles.* — Instead of fixing room dimensions, ceiling heights, thicknesses of walls, types of utilities, etc., housing codes should aim rather at establishing guiding principles, and the standards of performance required from a dwelling to be healthy, safe and convenient; such standards, in the absence of suitable scientific information, may best be related to good traditional building, critically evaluated.

4. Standards may serve the following two purposes:

a) They may be used as the basis on which dwellings are designed by housing agencies;

b) They may be used to control private builders, building for their own occupation or for others.

5. *Building by a public agency* — When a housing agency is directly responsible for housing, the standards adopted may be defined afresh for every project (being standards through experience). When housing activities are delegated to local agencies, the central agency may set standards or also approve type designs. Standards then merge into design.

6. *Building by private owners* — When house-building is for home-owners, the type of building and the accommodation provided will naturally depend on a family's resources; they may be augmented by gifts or loans, in cash or kind, or by technical assistance. However, some minimum type dwelling will be adopted. It may take the form of a «minimum-acceptable» demonstration house; or actual construction of a plinth. Areas may be set aside when a more temporary type of construction is permitted. With general house building, for practical purposes, the specific way in which housing principles and perfor-

mance standards have to be applied at a particular place and time will have to be defined in precise terms. Such definitions must always be regarded as provisional, being subject to revision within a short period, say not more than every five years. In view of the increasing application of scientific knowledge to building problems, the compilation of model codes may best be undertaken by a central authority competently advised; where necessary, these may have to be modified by local building authorities to suit local circumstances.

7. *Priorities* — Among the more important guiding principles in determining housing standards, the seminar would suggest the following:

a) Each dwelling should have individual sanitary facilities; though a separate bath-house is desirable, in some circumstances bath-house and latrine may be combined.

b) Where a piped water supply is available, the relative advantages of a separate supply for each dwelling — with reduced maintenance of fittings, wastage of water, etc. — should be balanced against its higher initial capital cost. Where no piped water supply is available, there should be provision for storage of water under hygienic conditions.

c) The aim of house design should be to provide at least two living rooms, in addition to space for cooking, so as to ensure privacy for the family. In some circumstances this may not at present be possible. House designs should, however, provide for expansion through stage-house plans.

d) Living spaces should be defined by a suitable floor area, based on the size of families mostly to be housed, rather than by specified room sizes, as this will give greater planning freedom.

e) House plans should always provide for the maximum use of screened and, where necessary, shaded open space either in garden, rooftop, courtyard or verandah. Such space is cheaper to provide than an enclosed room and may be equally suitable. House plans should provide storage space for such items as food, clothing, working tools and cycles; with some ingenuity, this space can be provided at little or no extra cost.

APPENDIX 2

BUILDING RESEARCH AND SIMILAR INSTITUTIONS CONCERNED WITH TROPICAL HOUSING

(The following lists some of the principal building research and similar institutions concerned in part or whole with tropical housing)

Building Research Station (Colonial Liaison Section), Garston, Watford, Herts., England.

Bureau Central d'Études pour les Équipements d'Outre-Mer (Service de l'Habitat et de l'Urbanisme), 90 Boulevard Latour-Mauborg, Paris 7e, France.

Central Building Research Institute, Roorkee, U.P., India.

Centro Interamericano de Vivienda, Apartado Aereo 6209, Bogota, D.E., Colombia, S.A.

National Building Organisation, 11-A, Jan Path, New Delhi, India.

National Building Research Institute, P.O. Box 395, Pretoria, South Africa.

Regional Housing Centro, Lembaga Penjelidikan Masalah Bangunan[2], Djl. Tamansari 124, Bandung, Indonesia.

West African Building Research Institute, P.O. Box 183, Accra, Ghana.

CONTROL OF ROOM CLIMATE IN TROPICAL AREA BUILDINGS

W. R. WOOLRICH

INTRODUCTION

Subsequent to World War I, both Europe and America experienced prophecy that future world leadership would fall to those who lived in cool climates. For more than a century the dominant political and economic power had been vested in people resident in the temperate zones, primarily those continents inhabited by fair-complexioned people. On the North American continent some political leaders took this pronouncement so seriously that they invited settlers to their cold climate lands with emphasized assurance to the migrants that, in keeping with the published prophecy, their offspring were destined to command national and world leadership. Unfortunately, some editors of highly reputable magazines with world circulation were among the gullible who joined in the broadcasting of this philosophy.

Historically, European and North American cold-climate people have been very successful in building a high level dominant civilization possessing remarkable energy and demonstrating successful leadership qualities. The past three decades, however, have produced vital changes in living conditions in many hot climate regions of the world, and these have significantly affected leadership trends. By the impact of the work of the medical and engineering professions, tropical diseases such as yellow fever, dysentery, typhus, and typhoid fever, malaria and hook worm have been rapidly placed under control. Vermin and pest disease carriers have been methodically reduced in number. Improved preservation methods have reduced hot-climate-induced food contamination to a new low.

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 721-733, 1959

As a direct result of the contributions of the medical and engineering sciences, there are millions of blond race people who have discovered new reserves of energy in regions included between the latitudes bounded by the 20°C (68°F) isotherms north and south of the equator. This is the area of the earth that engineers, physicians and meteorologists recognize as tropical in its thermal environment. Figure shows the extent of the area between these isotherms.

Within this zone may be found areas representative of both the hot-dry and the hot-humid climate. Again, these two major classifications may be subdivided into hot-dry areas with lowland and upland elevations, and hot-humid areas with mainland and with ocean island environments.

THE HUMAN BEING AS A HEAT MAKER

Heat comes from the body of a man primarily by evaporation, convection and radiation, although small percentages may be transferred by conduction. Usually if he is in an enclosed room, his body temperature of 98.6°F is higher than that of the air and the objects about him. This holds true even in summer periods. However, his skin temperature may be several degrees cooler than his body because of the rapid losses from the exposed surfaces by evaporation, convection and radiation.

When the ambient air is very still about the body, the film or layer next to the perspiring surface becomes highly saturated with water vapor and with the passage of time this air stagnation permits the thickness of this layer of high moisture saturation to increase. Conversely, as the air next to the skin surface is reacting saturation, the evaporation from the body approaches nearer and nearer to zero. This causes great discomfort to the individual.

Any small convection or movement of the air under the above conditions will break up this layer of humid air next to the body and improve the body evaporation with the result of a cooling of the body skin.

If the condition exists that this perspiring body with its envelope of saturated air is near to an impervious wall, a condition quite typical in a closed room of still air, a high concentration of vapor about the body is very noticeable to sensitive individuals. This sensitivity will

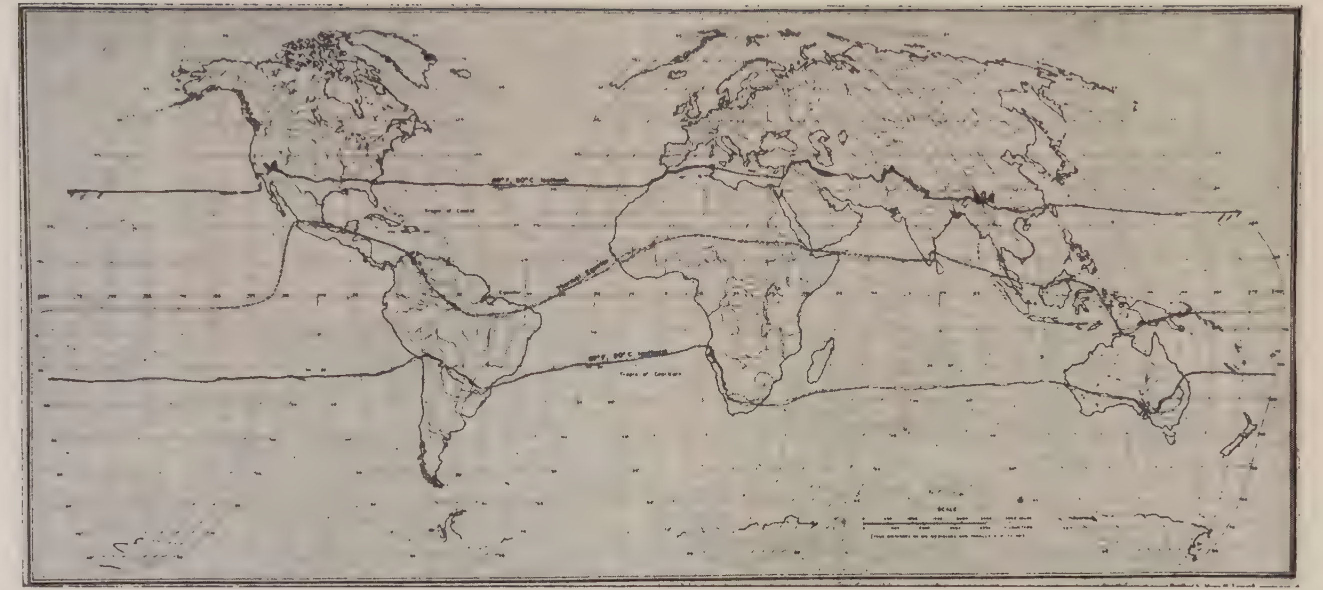
68°F, 20°C
Tropic of Cancer
Thermal Equator
Equator
68°F, 20°C
Tropic of Capricorn
SCALE

Fig. 1 — 20°C (68°F) isotherms — North and South of the Equator

be a function not only of the high humidity but also of the radiant heat energy exchange, the pressure of air compaction against the wall and the odor effects. Blind persons acquire a very real sense of detecting wall obstructions by such highly developed sensitivity which is probably made possible by those combined factors.

The radiant heat effect contributing to this degree of sensitivity is greatly affected by proximity to a wall surface having temperatures above or below that of the exposed body or, in case of fully dressed individuals, by the insulating clothing next to the skin. The transfer by radiation varies as the differences of the fourth power of the absolute temperature of the body and of the wall.

CLIMATIC CONTROL

Physiologically, the crux of climatic control is to maintain a suitable heat balance within each human being under consideration. The four climatic elements that affect man's bodily heat loss or gain are *temperature, humidity, air movement* and *radiation*.

The heat control facilities within the human body are very sensitive, and by virtue of the cooling and heating factors being opposed to each other, the internal temperature of healthy individuals normally varies but little from 98.6°F. These factors include on the contribution side the normal processes of metabolism, the absorption of different forms of radiant energy, and the convection-conduction heat transfer from air and from surrounding objects above body temperature. Factors of the opposing nature are radiation to colder wall surfaces and to the sky, heat transfer by convection-conduction to air and objects below body temperature, and the combined evaporation cooling effects from respiration and from skin perspiration. Within the body these processes are associated with a controlled variation in the rate of blood circulation near the skin surface when the surrounding air temperature changes. This physiological process serves as a compensating device to either retard or increase the body heat loss as may be needed for comfort.

VARIATION IN HUMAN THERMAL REACTIONS

Man's creation is believed to have taken place within an area where the mean annual temperature was above 20°C (68°F). Even today mankind does not enjoy room temperatures that drop below 14°C (57°F) or that soar much above 29°C (84°F). This is true whether the individuals originated from the races of the frozen north or from the equatorial regions.

The discovery of fire and the development of shelter and clothing made possible the migration of early human families toward the temperate zones with reasonable success. These transplantations in some cases of entire races, were effective in advancing the well-being of these peoples. The colder climate did something more — it changed millions of people from a heritage as nomads to a permanent-residence-loving people interested in vocations, avocations, education, churches, libraries and community life with permanently established cultural facilities.

We should not overlook the fact, however, that many of the millions of the people, whose progenators remained behind and established the civilizations of the past centuries within the hot-climate areas, have made real contributions to posterity on how to live with some control of heat stress in humans resident in such areas. They have accomplished this by the proven experiences of original design to meet their indoor climate cooling needs during seasons of excessive temperature. Many of the current generation of hot-climate people might have emerged in 1958 with as high an accomplishment quotient as that acquired by their temperate climate competitors, had their scientific insight of disease control and sanitation kept pace with their ability to avoid such heat stress by their elemental methods of designing clothing, buildings and diets to maintain a heat balance within their individual bodies.

Disease has exacted a frightful toll for scores of generations of the people of most hot-climate nations. Their infection with lingering diseases has not only lowered the individual energy and initiative of the residents in many of the tropical regions, but the *per capita* death rates have been excessive. Africa, Central and South America and

Asia each continue to have millions of people living under community conditions that are unsanitary, hazardous and vermin infested. Much of their water and food supply is unsafe for human consumption. Numerically, there are more people on this globe that are refugees from heat for more months per year than there are refugees from cold for a similar period, and these are too often the victims of tropical disease. People in cold climates can live more carelessly in matters of food and water handling because of the lower incidence of fatal contamination when low temperature storage is available. The increased rate of decay, brought on by high temperature ambient air, accelerates the rate of bacterial growth in nearly all perishable foods or liquids. On the other hand, the evidence that people living in warm climates are not destined to lives of low productivity when disease and unsanitary conditions are controlled is demonstrated by the millions of people with fair complexions now living south of the 20°C (68°F) isotherm of the United States under both hot-humid and hot-arid conditions, yet maintaining a high energy and low *per capita* death rate level equal to those in the temperate zones of their sister states. This change has been wrought within the past fifty years through the medical control of disease and by rigid enforcement of sanitary methods of perserving food, treating water, destroying vermin and insects, and designing homes.

GENERAL FACTORS RELATED TO INDOOR CLIMATE CONTROL IN ALL TROPICAL ZONES

Whether the area considered is in the hot-humid or in the hot-dry zones, certain factors will be of concern for both. These include orientation, solar heat control and the mass effects of walls.

ORIENTATION

Building orientation in shadeless tropical areas for solar protection will be most satisfactory on either side of the equator if the long axis of buildings are oriented on an east-west line. The principal objective of orientation should be to provide minimum direct sunlight on external walls of the inhabited space with maximum utilization of prevailing breezes for cross-ventilation in the warm seasons and maximum utilization of the sun's heat with full protection from the prevailing winds in the cold season. Even in very simple structures, capturing the prevailing breeze can be aided by orienting the structure for maximum cross ventilation in keeping with the maintenance of maximum protection against tropical rains and windstorms.

SOLAR HEAT CONTROL

In shadeless tropical areas, besides building orientation, the design of the roof, the walls, the doors and the windows are factors in solar heat control.

Unventilated, uninsulated pitched roofs can result in attic temperatures of 60°C (140°F). If such attic temperatures prevail, the ceillings in the room below will approach temperatures of 55°C (131°F). Since this ceiling heat will be transmitted largely by radiation to any individual beneath with a probable skin temperature of 30°C (86°F), such a person will be most uncomfortable and unproductive on account of this radiant heat stress.

Simple pitched roof homes with no room ceiling may be even more uncomfortable. Tests made at Austin, Texas, showed sun-exposed black and red roof surfaces of 85°C (185°F) on clear summer days. The under surface of such roofs may approach a mean temperature from 70°C (158°F) to 80°C (176°F) depending upon the roof structure and material. The radiant heating of the individual in such a room will far exceed the temperature of comfort and work productivity.

Roof shading, low conductivity roofing material, insulation and painting of the roof with reflective paint will greatly reduced the solar heating effects on the roof. Beneath the roof, adequate ventilation should be provided so that the room ceilling need not rise to a temperature above that of the prevailing ambient air in the adjacent shaded areas out of doors.

Roof construction, likewise, should be well ventilated by providing an air space between the roof and the ceiling. On account of the resistance to air movement in a horizontal enclosure under a flat roof, it is very desirable to insulate the ceiling beneath a flat roof adequately to maintain it at a reasonable temperature and, in turn, keep the temperature comfortable in the room beneath.

In areas near the equator, sunlight through windows on the equator side of building can cause great discomfort since the sun's inclination in the fall at these latitudes permits a marked solar heating load to enter unshaded openings on either side of the equator.

Those who live north of the 40th parallel in the northern hemisphere or south of the 40th parallel in the southern hemisphere are not greatly concerned with solar radiation on the southern and northern exposure of buildings respectively. In these temperate zones, by the time the sun has travelled appreciably toward the equator, in each instance the seasonal cooling winds offset the solar heating from the sun and residents may even welcome the solar increment of heat from the south (or north) exposure.

But at latitudes south and north of the 40th parallel, there is a decreasing influence of the seasonal cooling winds of fall as you approach the equator. By October and November in the northern hemisphere, and April and May in the southern hemisphere, the solar impingement on walls and windows on southern and northern exposures respectively, is a significant factor in raising the temperature in the tropical home. The use of glass windows for these exposures for buildings so located geographically should be limited to actual needs of observation and ventilation, the same as for eastern and western exposures.

MASS EFFECT OF WALLS

Some tropical-zone people by centuries of hard experience have learned to use the mass of walls having a high specific heat in an advantageous heating-cooling cycle. The mass is used for the storage of cold at night by the internal circulation of air, and it is used to keep the occupants cool during the following day by closing all openings to outside air infiltration. Usually the sleeping quarters of such homes are not inside the sun-heated walls, but are either out of doors within light latticed or screened structures, or on the roof or in the patio where they can take advantage of interstellar radiant cooling. By diurnal temperature changes, typical especially of hot-dry climates, the massive walls are cooled at night by air circulation through open windows and doors. During this cooling process not only are the lowered wet and dry bulb temperatures available for cooling the mass of the walls, but the roof is exposed to sky radiant cooling which often results in a temperature several degrees below the ambient wet bulb.

In both hot-humid and hot-dry climates, ceilings are most desirable for every room, and they should be designed to permit free ventilation between the ceiling and the roof to maintain temperatures therein not appreciably above that recorded by the ambient shade dry bulb thermometer.

For sleeping rooms, the design should be directed toward quick cooling of the space in order to more nearly approach outside night air conditions. This would indicate that this portion of the house should have walls that can be quickly cooled at night by either fan or natural circulation. More ideally, a screened-in space protected from rain and wind is to be recommended for low cost housing.

Since wire screening often does not provide either privacy for individuals, or protection of the space from winds and rains, some advantages can be obtained in designing walls with hollow spaces within them through which the night air can be circulated by fan to quickly reduce the accumulated daytime solar heat. This will apply equally to wood, brick, tile or stone construction. The effect of mass in retaining heat in different types of walls with natural ventilation is shown in Table I.

Let us now examine some of the characteristics of different types of wall construction. Assume a room having four walls, each fifteen feet long and ten feet high. The roof and floor of this room are constructed of perfect insulators so that no heat enters or leaves the room by these surfaces. If seven different types of construction are used, as indicated below, a very interesting comparison can be had as shown in the following table.

TABLE 1 — *Relative Time to Cool a 10' × 15' Room with Different Wall Mass*

Wall number	1	2	3	4	5	6	7
Wall area, Sq. ft. = 600	10" brick cavity wall	10" brick and clay tile	10" tile cavity wall	4" brick veneer on frame	Standard frame lumber	18" adobe solid clay	1" cork board
* "U" Value Btu/Ft² hr °F	0.34	0.29	0.25	0.28	0.26	0.34	0.34
Sp. heat Btu/lb. °F	0.200	0.182	0.150	0.250	0.450	0.180	0,480
Wall weight, pounds	49,800	39,000	27,000	31,200	12,000	103,500	250
Thermal energy at △ T = 10 °F	99,600	71,000	40,500	78,000	54,000	186,000	2,100
** Time to cool 10 °F, hrs.	24.4	20.2	13.5	23.2	17.3	45.6	0.3

* This value includes the air film coefficient on each side of the wall.

** Calculated on the assumption that inside air temperature is held at 80°F, that outside air has drupped to a constant temperature of 80°F and that the "U" values remain constant as given in the table.

CONTROL OF THE INDOOR CLIMATE IN THE HOT-HUMID TROPICAL ZONES

In the hot-humid tropical zones, while the dry bulb temperature of the ambient air may be lower than that for a similar hot-dry area, the wet bulb temperature that prevails both night and day will be uncomfortably high. In this environment, the most effective and inexpensive method of reducing thermal discomfort is to provide air movement over the skin to speed up evaporation.

Even in very simple structures, capturing the prevailing breeze can be aided by orientation for maximum cross ventilation in keeping with maintaining maximum protection against rain and windstorm. Such effective cross ventilation does require provision within the room against dead air zones often occasioned by high window sills, and also from ceiling air pockets from which the hot air cannot escape. Natural ventilation of such space can be aided greatly by simple vertical window scoops, a judicious use of full opening casement windows and ventilating exit air openings in the high points of any room. The use of movable screens in lieu of permanent partitions has the advantage of permiting oriented breeze deflection with a very light material mass.

CONTROL OF THE INDOOR CLIMATE IN THE HOT-DRY TROPICAL ZONES

The use of heavy unshaded walls in the hot-dry climate of buildings not air-conditioned should be avoided when structurally possible. These walls absorb large quantities of heat that will not be readily cooled without special treatment. A more acceptable house design for this type of climate would be an inside structure of heavy walls to carry the structural load and relatively light weight outside walls serving only as space enclosures. When the sun has gone down this heavy inside mass that has been little effected by the solar rays during the daytime can be continuously cooled throughout the night by the circulation induced by attic fans pulling the night air over the mass, thence to the attic and out.

This method is effectively used by many designers. The usual procedure on the following morning is to close off the fan when the outside dry bulb temperature rises above the inside room temperature, then close up the living room space to prevent infiltration of warmer air than that existent within the rooms.

When a daytime fresh air supply is essential from the outside, the dry bulb temperature can be considerably modified by admitting the incoming air from over verdant shrubs and bushes. The high rate of transpiration of fully leaved bushes gives an evaporative cooling effect and may moderate the incoming air dry bulb temperature from 3 to 8 degrees centigrade.

Evaporative cooling in itself is most satisfatory in hot-dry climates. In single-stage evaporative cooling, room temperatures within 2 to 3 degrees centigrade of the ambient wet bulb temperature may be maintained. If two-stage evaporation cooling is used, temperatures below the wet bulb temperature are quite possible and may approach within 2 to 3 degrees centigrade of the dew point. For example, in an area where midday dry bulb temperatures soar to 110°F, but the wet bulb remains about 20°C (68°F), stage evaporative cooling to 10°C (50°F) is practical.

ADAPTATION OF LIVING SPACE TO REFRIGERATED COOLING

Refrigerated cooling is especially effective in hot-humid climates, due to its dehydrating capacity with its cold condensing surfaces. The cooling coils may condense many liters of excess moisture from the air per day and give a resultant atmosphere that is drier and healthier for the individuals inside.

Advances have been made within the past five years that make the water-cooled condenser unnecessary in capacities up to twenty tons of cooling capacity, even in hot climates. The greater power demand to operate an air-cooled compressor is largely offset by the higher cost of pumping and maintaining a water cooling system with liquid cooled condensers and associated cooling towers.

House designs for machine cooling differ from those for natural or fan cooling in that the outside walls and ceiling should be adequately insulated to keep the heat infiltration and conduction as low as possible.

RADIANT COOLING OF ROOMS

Considerable success has been experienced in the United States with the radiant cooling of homes. Filtered dehumidified and refrigerated air is delivered to the living space which is kept under a slight pressure to prevent infiltration of outside air.

INCREASE OF PERSONAL PRODUCTIVITY IN COOLED WORKING AND LIVING SPACE

Currently, accurate statistics are not available on the increase of productivity with the moderation of interior climate in either homes or factories, although general observations indicate marked improvement in human efficiency. Extensive studies are planned by our universities and engineering societies to determine the economic value of the air cooling of hot climate living and working spaces. Until such studies have been completed, the value of cooling must be predicated on these many observations that give a relative preference to a working environment where the human stress of both heat and cold is held to a minimum.

GENERAL DISCUSSION

Dr. G. P. Crowden (U. K.) — Referring to the importance of ventilation as emphasized by Dr. Fraser he said that the heat given out by an adult in one hour could raise the temperature of 600 cubic feet of air by 10°F or more and that this indicated how necessary it was to provide air change to remove heat from living rooms during occupation.

Referring to the paper by Mr. G. A. Atkinson: he said he would welcome the views of the speaker on the use of corrugated aluminum and corrugated for roofs in tropical dwellings.

Dr. W. V. Macfarlane (Aust.) — After Dr. Atkinson account of mass apartments in the tropics of wondered whether there were data on: 1) the wet bulb temperatures in the rooms; 2) the air flow through the rooms; 3) the effect of flat roofs in the thermal environment of the upper floors of the buildings.

Mr. G. A. Atkinson (U. K.) — My comments are directed to four matters:

i) the importance of night-time conditions in low-cost housing;
ii) ventilation, particularly in equatorial («doldrums») climates;
iii) the insulation of metal roofs.

i) *Night-time* conditions. Especially in upland areas and close to the Tropics temperatures at night in the cooler season commonly drop the low 70°F's and below. Then, at least in poorly insulated dwellings of lightweight construction such as metal sheeting on a frame or thin concrete panels, cold at night may be the critical condition especially with poorer families unable to afford proper bedclothes. To keep warm, the rate of air change is restricted by closing windows and blocking up ventilators. Where night temperatures fall sharply, the closing of openings may not be enough in poorly insulated houses. The resulting cold may cause sleeplessness, or cause occupants to use possibly obnoxious open fires in their bedrooms. Problems of carbon monoxide poisoning from open braziers in African dwellings are causing concern in South Africa. C. G. Webb, Building Research Station, in a paper to be published on the thermal design of bedrooms for tropical low-cost housing, has suggested that there is a definite value of the conduction loss from a thin-walled house which should not be exceeded. Under tropical conditions, this value is related to the number of occupants. The paper shows how to determine the value graphically.

ii) *Ventilation, particularly in «doldrum» climates.* Crowden has mentioned the important role of ventilation in removing excessive heat due to thermal conduction and storage by the structure and to the metabolic heat of occupants. C. G. Webb (Journal of the Royal Institute of British Architects, November 1957) has put into graphical form the equations for the stack effect which is the principal manner in which this excess heat is removed under still air conditions. He has suggested a ventilation index and a suitable scale of ventilation. These should serve as a guide to designers of tropical housing. Building climate data, mainly from Singapore, is being analysed; further data, particularly from very crowded housing like that described in Hong Kong, would be welcome.

iii) *The insulation of metal roofs.* Crowden asks how thermal conditions under corrugated sheet steel roofing can be improved. «Corrugated iron» is popular in cheap tropical housing because of its price and availability; aluminium roofing, though thermally an improvement, costs more. An alternative, meriting greater use, is to drape paper-backed aluminium foil under steel sheets.

It provides an air space as well as reflective insulation and so is advantageous for both day and night conditions; it should not be costly.

2ND SESSION: **Industrial hygiene** — *Friday, September, 12th*
11:00 to 12:30 a.m. Room B

The meeting was held under the Chairmanship of Dr. Cornelis de Langen. The Rapporteur was Dr. Ronald K. Macpherson and the Secretary was Dr. Mário A. de Mendonça e Silva.

The following papers were presented and discussed:

INDUSTRIAL HYGIENE IN THE TROPICS

R. K. MACPHERSON

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The title of this paper is not to be taken to imply that there are two kinds of industrial hygiene — industrial hygiene in the tropics and industrial hygiene in the «not-tropics». That is not so. The essentials in the practice of industrial hygiene in the tropics do not differ from those in temperate climates, they are merely modified in certain directions determined by the climate, the people and the industries concerned. But even in respect to these modifications the differences are not as great as may appear at first sight.

Even those occupations which by their very nature can occur only in tropical climates are found on analysis to present problems essentially the same as those of industries in temperate climates. The handling of pineapples and sugar cane requires standard methods for the protection of eyes and hands from injury (Holmes, 1956), and the sequence of events in bagassosis — an allergic reaction leading to bronchiolitis and later emphysema and bronchiectasis (Ganguly and Pal, 1955) — are those commonly associated with the inhalation of irritating dusts. Similarly, the effects of silicosis of the lungs are the same whether they result from the manufacture of pottery in Staffordshire in England or from mining granite in Singapore (Khoo, 1957).

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It can therefore be said that basically the industrial hygiene of the tropics is the industrial hygiene of temperate climates. Having made this point, however, it is instructive to consider the effect of the modifying factors mentioned — the climate, the people and the type of industry concerned.

Climate is undoubtedly the most potent single influence which determines the particular nature of industrial health problems in the tropics. The necessity for the provision of an acceptable thermal environment for factory workers has long been recognised in temperate climates. It is well established that excessively hot conditions affect not only the welfare of the workers but also the quantity and quality of the output. This point need not be further elaborated as the subject has recently been comprehensively reviewed (Bedford and Tredre, 1955).

It is also true, unfortunately, that in tropical climates, where the stress of hot industries is greatly increased, insufficient attention has been paid to this important point. There are many reasons why this is so. Perhaps the most popular and most fallacious is that the indigenous inhabitants of hot climates are capable of withstanding higher environmental temperatures than the white races of the temperate zones. There is a growing body of evidence against this view but it is sufficient to quote one very plain statement by Ladell (1955) based on observations on men working in a West African gold mine — «Nothing was found to indicate that the West African working in a deep mine has an unusually high heat tolerance».

Although in temperate climates the necessity for heating of factories during winter is readly accepted, it is often difficult to persuade factory management in tropical areas to take the logical converse step of employing air conditioning in the summer. The most convincing argument is, of course, to show that output will increase, but investigations designed to this end often meet with unexpected difficulties. For example, an enquiry into environmental conditions in flour mills in India (Subrahmanyan and Majumder, 1953) showed that the controlling factor in output was not environmental conditons but the government quota of wheat. However, there is a growing awareness of the importance of environmental conditions in factories and workshops in the tropics. An extensive study has recently been made of such conditions in Indian textile mills (Ashe, Largent and

Roberts, 1957) which with its recommendations may well serve as a model for other investigations of a like nature.

Hot conditions may be important in a more indirect fashion than that just described. The basis of many measures to protect workers from industrial hazards is the wearing of protective clothing. The very fact that such clothing is protective usually means that it is stout and impervious, so that the wearing of it in hot conditions may not be merely disliked by workers but may be physically impossible.

Scanty clothing and a skin wet with sweat provides an ideal combination of conditions for the absorbtion of toxic substances by way of the skin. It would seem also that high environmental temperatures may intensify the action of toxic substances. Baetjer and Smith (1956), for example, have investigated the effect of environmental temperature on the reaction of mice to parathion and have shown that at 96°F the mortality was higher that at 73°F. Sweating may however, in rare cases, confer an advantage. Shiels (1954) has shown that large quantities of lead may be excreted in the sweat and this may be the reason for the observation made in Australia (Shiels, 1955) that the incidence of plumbism was less in a warmer than in a cooler environment where other conditions appeared to be identical.

Perhaps even more important than the climate in determining the special nature of industrial health problems in the tropics is the nature of the people concerned, because it must not be forgotten that the problems of industrial medicine are first and foremost the problems of men and women and only secondarily those of physics, chemistry and biology.

It must be accepted that in many places in the tropics low standards of health, nutrition and education prevail with important consequences in industry. De Briey (1955) makes the point that poor physical health is largely responsible for the low output of African labour. This is probably equally true in other areas. De Briey goes further, however, and stresses the significance of the psychological impact of a new industrial way of life on Africans whose old traditions and cultures have in the past sustained them effectively.

The industrial revolution in Europe has been relatively slow. There has been time for adaptation on the part of the workers to the new order. Sufficient time has elapsed for the growth of an industrial tradition which imposes standards of conduct for workers and employers alike and for the development of a code of industrial legislation which is accepted or, more rarely, enforced unhesitatingly. By comparison, the industrialization of the tropics has been a whirlwind affair and industry as a whole lacks these standards of behaviour. The workers may be ignorant of the provision of industrial legislation, or it may be evaded by employers.

There has been time in Europe for appreciation by all workers that any type of machinery may be dangerous and that any industrial process may prove to be injurious to health. Native populations have none of this folk-lore of tradition to guide them and the dissemination of knowledge of these matters is of itself one of the great problems of industrial health. It is difficult to convince a native worker that he must wear protective clothing, that he must wash his hands before meals, and equally difficult to devise adequate warning notices for workmen who are illiterate. Progress is, however, being made. Considerable attention is being devoted to health surveys in industrial communities in the tropics (e.g. Cambon and Cambon, 1955), and to the physical preparation of the native labourer for the task he has to perform (Wyndham, Strydom, Morrison, Du Toit and Kroon, 1954) and to the general problems of human biology as they apply to workers both native and European in the tropics (Vigan, 1956; Macpherson, 1956).

It is not to be thought that the problems of industrial health in the tropics are confined to the indigenous inhabitants. The employment of Europeans in hot climates may present special difficulties. When isolation and boredom are added to the effects of a hostile physical environment the result is often inefficiency and unhappiness or even, in some cases, incapacitating psychological mal-adjustment.

Many tropical countries are experiencing the impact of a modern industrial revolution on an economy which was previously based on agriculture or small-scale industries. Much depends in these cases on the circumstances in which the new industries are introduced. Critchley (1955), in discussing the health of the industrial worker in Iraq, emphasises the value of the high standards set by the Iraq Petroleum Company.

The third factor concerned in determining the special problems of industrial hygiene in the tropics — the type of industry concerned — has of necessity been discussed in part in the previous sections and may therefore be dealt with more briefly. It is perhaps customary to associate health problems with the industrial towns of temperate climate and to think of tropical industries as being largely agricultural and hence as devoid of risks to the workers. This is, of course, by no means true. Many industrial practices, such as mining, have long been established in tropical areas and many tropical countries, for example India, have long been manufacturing centres. Furthermore agriculture, no less than urban industry, has its health problems. As Stott (1954) has said, «one is inclined to forget that occupational disease can and does occur in the country». Even the cultivation of rice (Pancheri, 1956) has its industrial hazards, and the handling of crops such as grain can produce serious dust problems, as the International Labour Office (Occupational, Safety & Health, 1956) has shown.

Nevertheless, whilst bearing in mind that generalizations by their very nature are apt to be inaccurate, it may be said that tropical countries as a whole are still the main producers of «commodities», that is to say the products of agriculture and mining, and that manufacturing industries are of lesser importance. There are, however, two changes occurring simultaneously — a growing importance of secondary industries and an increase in the scale and the mechanization of the older primary industries. Both these changes have introduced new industrial hazards, and this is particularly striking in agriculture.

The use of machinery by unskilled workers can lead to disabling injuries, but the risk is usually comprehensible even to the unsophisticated. The growing use of chemical pesticides has introduced into agriculture industrial hazards of a most insidious and dangerous kind. The importance of these risks is emphasised by the increasing attention being devoted to them (Barnes, 1953; Edson, 1955; McGee, 1955; W.H.O., 1956; McQueen, 1956). These pesticides are of especial importance in the tropics because in addition to their use in agriculture they are being used on an increasing scale in the control of disease vectors.

These matters are to be dealt with in a special paper in this session, and in consequence it would be very wrong of me to discuss them at any length. There is, however, one point that I would wish to make. Menon (1958) has recently reported the occurrence of nine deaths in Borneo from the use of pentachlorphenol. At first sight his paper seems to emphasise that the special problems associated with industrial hygiene in the tropics are due to the nature of the population at risk. The victims were primitive people, Sea Dyaks (Malays and Chinese were not affected), they were undernourished and of poor physique, suffered from helminthiasis, wore no protective clothing, did not bath regularly and did not wash their hands before meals. Strangely enough however, Gordon (1956) has also reported nine cases of pentachlorphenol poisoning occurring among agricultural workers in Queensland, Europeans who, presumably, were neither primitive nor undernourished, but they did have this in common with the Borneo victims — they were working in hot conditions. This brings us back, I think, to the point that the effect of climate is the dominating factor in determining the special nature of tropical industrial hygiene.

Sir Charles Martin, some thirty years ago (Martin, 1930), said «Work in hot surroundings demands a stout heart». All those concerned with welfare of workers in the tropics, whether European or indigenous, should remember that this is both literally and metaphorically true.

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CONTROL OF ORGANIC INSECTICIDAL HAZARDS IN THE TROPICS[1]

HENRY N. DOYLE[2]

A major challenge of the chemical industry in the past century has been the development of insecticides that would supplement the natural ones and at the same time overcome their shortcomings. By the late 19th century, man's continuing progress had become increasingly dependant upon the discovery of economic poisons with prolonged residual effects and highly lethal properties. In response to this need, inorganic chemistry, in its zenith at that time, developed arsenicals and fluorides for use singly or in combination with the natural organic insecticides. These chemicals, however, only partially fulfilled the need for effective insect and pest control agents.

In 1943, a significant chain of events was set off in the field of organic chemistry, with the recognition of the spectacular insecticidal properties of DDT. A vast amount of research was stimulated, and by 1958 the Entomological Society of America listed 67 synthetic organic insecticides (1). In the United States alone the production of organic insecticides and rodenticides in 1956 amounted to over 330 million pounds, with a value of approximately $90 million (2).

This explosive growth of economic poisons within the past 15 years has not been without attendant problems. The particular problem of concern to this group involves the potential toxic hazards of the organic insecticides to man, arising both from their manufacture and application and from contaminated foods. Because of the broad scope of this subject, this paper is limited to the development of

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industrial hygiene controls in the application of insecticides, with special reference to problems of tropical areas. In view of the acute problem in the world-wide malaria eradication program, emphasis is placed in safety measures relating to the use of dieldrin.

Whether insecticides are more toxic to human beings exposed in tropical climates as compared to temperate climate is not generally known. However, certain environmental factors in the tropics mitigate against the use of proper protective measures. Foremost among these is the difficulty in wearing proper protective equipment under conditions of high temperature and humidity, and the natural reluctance of the tropical worker to be encumbered with excessive clothing. Since malaria occurs more frequently in rural areas, other problems are introduced, such as inadequate facilities for proper personal hygiene and the absence of medical centers for personnel selection, medical supervision of the worker, and treatment of the worker following first signs of intoxication. The lack of communications and highways in malarious areas is also a factor which prevents adequate supervision and causes logistical problems in maintaining spraying crews with proper protective equipment.

The toxicology and pharmacology of most of the commonly used organic pesticides have been well developed (3, 4). For the most part, the pesticides fall into three general chemical and toxicologic classes: 1) chlorinated hydrocarbons, which are central nervous system poisons; 2) organo-phosphorus compounds, wich inhibit certain enzyme systems of the body, especially cholinesterase; and 3) the dinitrophenols, which increase the oxidative metabolism and therefore the heat production of the body. Tables I, II and III taken from the *Clinical Memoranda on Economic Poisons* (3) list the more commonly used pesticides together with their oral and dermal LD_{50} values.

In the application of insecticides, dieldrin, a chlorinated hydrocarbon, and TEPP and parathion, organo-phosphorus compounds, have caused the greatest difficulty in man in the Western Hemisphere. Two factors account for this — their inherent toxicity and extensive use. Dieldrin came into general use as an antimalarial insecticide in 1952. Since then over 9,000,000 pounds of 100 percent dieldrin have been used in 55 countries for vector control purposes. Eight of these 55 countries have accounted for 2,000,000 pounds.

TABLE I — *Acute oral and dermal LD_{50} values of chlorinated hydrocarbon insecticides for male and female white rats*

Compound	Oral LD_{50} (mg/kg)		Dermal LD_{50} (mg/kg)	
	Males	Females	Males	Females
Methoxychlor	(6000.0) **	—	—	6000.0 *
Chlorobenzilate	1040.0 *	—	—	—
DDE	880.0 *	1240.0 *	—	—
DDA	740.0 *	—	—	—
Chlordane	335.0 *	430.0 *	840.0 *	690.0 *
Lindane	(200.0) **	—	500.0 *	—
DDT	113.0 *	118.0 *	—	2510.0 *
Toxaphene	90.0 *	80.0 *	2300 0 *	—
Dieldrin	46.0 *	46.0 *	90.0 *	60.0 *
Endrin	17.8 *	7.5 *	—	15.0 *
Dilan	—	—	6900.0 *	5900.0 *

* These values were determined at Technical Development Laboratories under standardized conditions.
** Sex not specified.

TABLE II — *Acute oral and dermal LD_{50} values of organic phosphorus insecticides for male and female white rats*

Compound	Oral LD_{50} (mg/kg)		Dermal LD_{50} (mg/kg)	
	Males	Females	Males	Females
Malathion	1375.0 *	1000 0 *	4444.0 *	4444.0 *
Chlorthion	880.0 *	980.0 *	1500.0 -4500.0 *	4100.0 *
Dipterex	630.0 *	—	—	—
Diazinon	108.0 *	76.0 *	—	180.0 *
Parathion	13.0 *	3.6 *	21.0 *	10.9 *
Demeton	6.2 *	2.5 *	14.0	8.2 *
TEPP	2.0	1.2	—	—

* These values were determined at Technical Development Laboratories under standardizet conditions.

TABLE III — *Oral toxicity of selected substituted dinitrophenols to the rat*

Compound (R = 4,6-dinitrophenol)	Tolerated acute dosage (mg/kg)	Acute LD_{50} (mg/kg)	Tolerated conc. in diet (ppm)
2-Methyl-R (DNOC)	10	30	100
2-sec-Butyl-R	5	37	100
2-Cyclohexyl-R	30	80	500

Illness in spraymen was first reported by Carrilo of Venezuela in 1954 (5). Other reports of human intoxication followed from Ecuador, Nigeria and India. Unconfirmed reports have also indicated difficulty with this insecticide in certain southeastern Asian countries. Hayes has described dieldrin poisoning in 63 of 417 spraymen exposed in Venezuela, Ecuador and Nigeria, with an attack rate varying from 9 to 18 percent, but with only one fatality (6). The clinical features of the cases reported by Hayes are quite similar to 20 cases of dieldrin poisoning previously reported by Patel and Rao in Bombay State, India (7). All of the clinical cases reported by Hayes were severe enough for the patients to seek medical aid. Half or more of the patients had convulsions.

The mild clinical illness caused by dieldrin was characterized by headaches, blurred vision, dizziness, sweating, difficulty in sleeping, bad dreams, nausea and general malaise. The more severe illness was characterized by the same symptoms and by jerking of the entire muscle groups, leading to movement of some part of the body or the limbs or even causing the patient to fall. In extreme cases, these movements were accompanied by loss of consciousness. The more severe non-fatal cases reported by Hayes involved one or more epileptic convulsions with loss of consciousness. It was impossible to establish the estimated dose received by those who became ill, but the earliest observed poisoning occurred after a little less than three months' exposure. In general, the proportion of sprayers poisoned increased with the duration of their work. Twenty-six percent of the workers exposed for eight months or more became ill.

In the United States, organo-phosphorus compounds, especially TEPP and parathion, have accounted for the majority of poisonings due to insecticides. Several deaths have been reported in orchard and agricultural workers. Transitory illness has been identified in aircraft pilots (8), and it has been suspected that several aircraft accidents might have been due to blurred vision of the pilot because of exposure to organo-phosphorus compounds. The drifting cloud following dusting with organo-phosphorus compounds has also been implicated in several deaths and illnesses, but this has not been confirmed.

The herbicides apparently have not been involved extensively in known human intoxications in the tropics, although the dinitro-ortho-cresols have caused several deaths in Great Britain (9). Because the symptoms of dinitro poisoning closely resemble those of heat exhaustion, it is possible that unrecognized cases may have occurred.

In designing protective measures for the sprayer, the industrial hygienist is faced with two unique factors. First, the insecticide is highly toxic through the dermal route. Second, the environment does not lend itself to control by the usual industrial hygiene methods of substitution, insolation and ventilation. Thus, protective clothing and respiratory protection become the fundamental methods of control.

When the insecticide reaches the sprayman, inhalation toxicity become minimal, while the dermal route assumes considerable importance. Only to the extent that the sprayman is involved in the mixing of the insecticide in its dry, powdered form, prior to spraying, is there some hazard through the inhalation route. The sprayman's exposure thus results almost entirely from the dermal route. The severity of this exposure is pointed up by the fact that the dermal LD_{50} for many of the insecticides, including parathion and dieldrin, is of the same order of magnitude as the oral LD_{50}.

The first difficulty arises in the mixing operation where the worker may have a slight exposure to dust and where, too frequently, the uninstructed worker uses his hands and arms to stir the solution. Following transfer to the pump, where some splashing on the lower extremities is difficult to avoid, the worker's next exposure is to a simulated rain where the mass median particle size of the aerosol is about 10 microns, too large to penetrate deeply into the respiratory system. The dermal rate of absorption for man has not been developed, but it is reported that in the case of toxophene, a single dermal application of 46 grams or a daily application of 2.4 grams over a period of days is very dangerous (3). Under the usual mixing and spraying conditions, it is apparent that this figures of daily exposure could be readily exceeded. Since dieldrin has a greater dermal toxicity for white rats than toxophene, it is reasonable to assume that an amount less than 2.4 grams would be dangerous. Reports on dieldrin poisoning due to spraying operation have noted the absence of adequate protective clothing.

Respiratory protection against the chlorinated hydrocarbon insecticides where the exposure is to dust, as in repackaging, formulation preparation, and in dusting operations, is desirable for the less toxic ones such as DDT, and essential for the more toxic ones, such as dieldrin. Since the chlorinated hydrocarbons, in general, have a low vapor pressure, a dust respirator, consisting of a half-mask and filter is sufficient.

The organo-phosphorus insecticides, however, have relatively high vapor pressures, and it must be assumed that their vapors are toxic to man. Thus, respiratory protection is required under all conditions of use. Since the exposure is to dust and vapor, a half-mask canister respirator fitted with an absorbent capsule of activated carbon is required, in addition to a particulate filter.

RECOMMENDATIONS BY W.H.O. COMMITTEE ON INSECTICIDES

Recognizing the health hazard inherent in certain of the newer insecticides, the WHO Expert Committee on Insecticides in October 1956 made some recommendations regarding health protection (10). The measures recommended were intended to apply to those insecticides presenting a somewhat greater hazard than DDT and BHC, and, as the Committee emphasized, they were not to be necessarily considered as adequate for the more toxic ones.

As protective clothing, the Committee recommended that spraymen wear a wide-brimmed impervious helmet and face shield. For toxic insecticides presenting a strong hazard through inhalation, a respirator was recommended. In temperate climates, it was recommended that the body be covered with a full-length coat or overall with high collar, long sleeves with tight-fitting wrist, and double-breasted fastenings. For tropical climates, a waterproof poncho-type cape sufficiently long to protect the legs should be provided. For hand and arm protection, gauntlet type rubber gloves (30 cm) with cotton liners were recommended. The legs should be covered with long trousers and boots, or brodequins should be worn. Weekly washing of the clothes under supervision was suggested. In addition, hygienic measures were suggested for safe mixing and spraying and sound practices for good personal hygiene, health education and medical control were outlined.

RECOMMENDATIONS BY W.H.O. STUDY GROUP ON TOXIC HAZARDS OF PESTICIDES

The WHO Study Group on the Toxic Hazards of Pesticides, in June 1956, reemphasized the toxicity problem and urged the various governments in cooperation with WHO to conduct environmental studies under varying conditions (11). The study group pointed out that information on the toxicity of insecticides will only give an approximate idea of the danger unless something is known about the quantities absorbed and the routes of absorption. More importantly, however, the Committee recognized the worker's difficulty in carrying out spraying when clad in full protective clothing and took cognizance of the findings of the Communicable Disease Center of the U. S. Public Health Service that exposure occurs to a very considerable extent by way of the skin, and that this route far exceeds that by inhalation. With this in mind, the study group suggested additional precautionary methods for all spray operations and outlined special precautions for dieldrin and the organo-phosphorus compounds.

However, sound as they are, the application of these recommendations under tropical conditions becomes a difficult task, especially those relating to protective clothing, since the worker by nature is accustomed to wearing a minimum amount of clothing for comfort reasons. Also, a worker shrouded in impervious clothing under conditions of high temperature and humidity can suffer a physiologic insult which may be of greater individual significance than the toxicity of the insecticide.

PAN AMERICAN SANITARY BUREAU COMMITTEE ON PROTECTIVE MEASURES

Special attention has recently been focused on intoxication problems associated with malaria control programs in Latin American countries. Nineteen Western Hemisphere countries have malaria eradication programs in which three to four thousand persons are employed, with an annual consumption rate of over 400,000 pounds of dieldrin, although dieldrin is not used by all countries. As a result of the continuing problem of dieldrin poisoning in these programs, the Pan American Sanitary Bureau late last year (1957) appointed an expert Committee on Protective Measures. The Committee has met once to date, its first aim being to develop protective measures for the application of insecticides of toxicity comparable to dieldrin. Proceeding on the assumption that the insecticides in general use in the malaria eradication programs, especially dieldrin, were more toxic through the dermal route, and using the report of the WHO Expert Committee on Insecticides (WHO Technical Report 110 and 114) as a basis, the group developed tentative guides for the protection of spraymen.

As a member of this study group, I should like to give you my interpretation of the Committee's conclusions. It was the consensus that certain modifications to the WHO report should be made, especially with respect to protective clothing. It was agreed, although not unanimously, that the sepraymen should be furnished with underwear, consisting of a T-shirt and cotton shorts, to provide a protective layer of cloth between the contaminated garment and the skin, as well as to absorb perspiration. In addition, the sprayer was to be provided with heavy ribbed cotton socks and a half-leather boot with a non-slip synthetic impervious sole.

In an attempt to avoid the use of an impervious covering, such as a poncho, the Committee considered coveralls with impregnated or reinforced cloth at the shoulders, sleeves and thighs, the areas of greatest contamination. However, several members felt that a poncho was necessary, especially during the actual spraying operation. It was finally suggested that there be provided a short cape extending about halfway between the elbows and shoulders, constructed of a light impervious material with a high tensile strenght and designed so that it could be tied around the sprayer's neck in much the same manner as a barber's apron.

The impervious helmet and face shield, as described by the WHO Committee, was continued, but the respirator was discarded since, in the group's opinion, the respiratory route of intoxication was of small significance. It was recommended, however, that the spraymen be provided with a gauze mask similar to a Martindale respirator. This mask was not expected to provide any protection to the respiratory system, but simply to prevent droplets and entrainment from entering the mouth or nose where further absorption might occur in the mucous membranes and by ingestion. The gauze would of necessity have to be changed several times a day.

It was recommended that all clothing be furnished the spraymen and that the clothing be laundered daily, in contrast to the WHO recommendation of weekly laundering. Since on many occasions the crews are removed from laundering facilities, it was recognized that such a procedure would be difficult at times. In this connection, it is interesting to note that Venezuela has recently adopted the policy of providing spraymen with six uniforms so that a clean one is available each day. The Committee further recommended that the spray crews be instructed to wear the protective equipment only during working hours.

The Committee also felt that inadequate attention was being given to personal hygiene and recommended that every effort be made to provide wash periods before meals and to have the spraymen bathe with soap at least once a day. It was the consensus that personal hygiene was the most valuable preventive measure.

The Committee stressed the importance of an educational program for the worker which would: *a*) inform him about the toxic hazards of the insecticide and emphasize that with proper safety procedures, it could be handled safely; *b*) instruct him in the proper use of his safety equipment and the reasons for its use, with particular attention to good personal hygiene; and *c*) instruct him in the proper and safe techniques of spraying. Also as part of this program, foremen and supervisory personal should be provided with educational material of a degree slightly higher than that furnished the spraymen, and instructed to recognize signs and symptoms of the disease so that workers could be referred for immediate medical aid.

It was also felt that the workday should be limited to eight hours and that a midday break for lunch and rest be provided.

Expressed needs were for the epidemiologic approach to be used in investigating insecticide intoxication, for applied research on various kinds of protective equipment adaptable to a tropical environment and for investigating additional protective equipment. Research on spraying methods which would minimize the exposure to the worker and improve on present spraying techniques was pointed out as being desirable.

WORKER EDUCATION

The importance of worker education cannot be over-emphasized, since the spray crews work frequently in remote areas where adequate supervision is not possible. At least one company has included in each package of insecticide a pictogram (figure 1) showing good and poor practices.

However, some diagrams showing good practices are contrary to the recommendations of the WHO and PASB committees in that the worker is not provided with gloves or coverings for his legs. Also, the hat shown in this pictogram is obviously a cotton one, which would absorb considerable quantities of the insecticide. The washing procedure, too, is inadequate, since it is limited to hand washing and fails to emphasize the need for daily baths. Although this is an excellent start in educational material for the worker, presented at his level, it is obvious that certain improvements could be made in these pictograms. Several countries have modified this basic idea to suit their own needs and have inserted appropriate warnings to call attention to the hazards in language which the worker can understand.

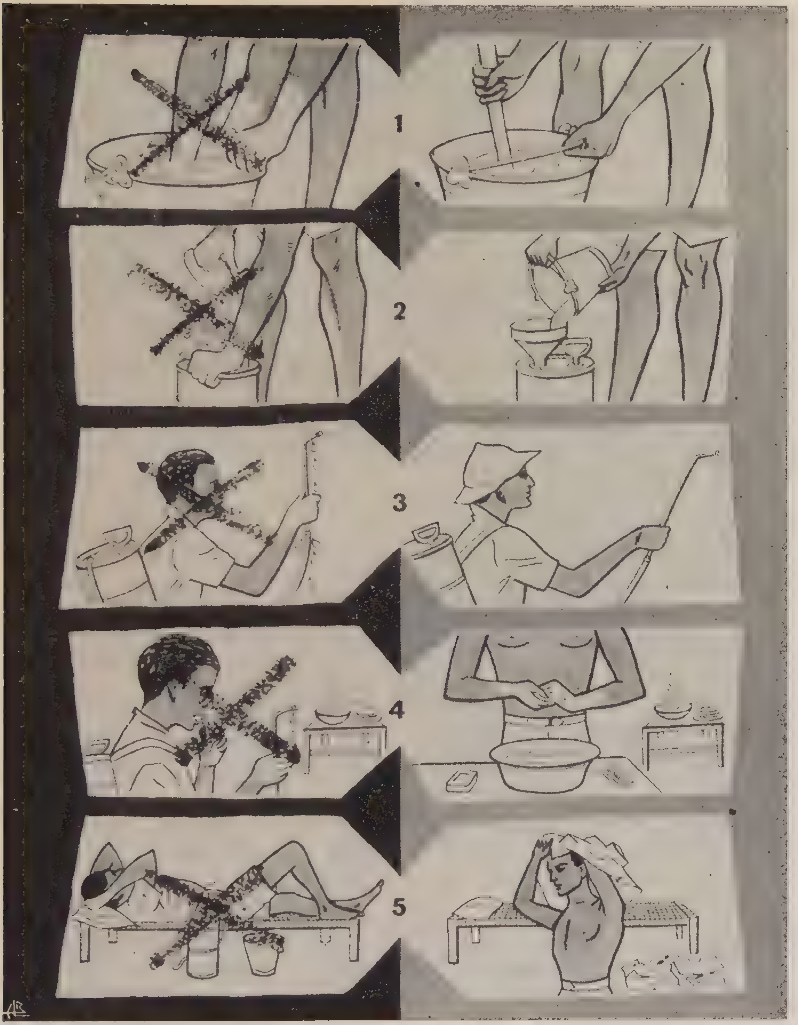

Fig. 1

SAFETY PROGRAM

Through the use of dieldrin and other toxic chemicals with superior insecticidal properties, man for the first time in history has an instrument which may completely eradicate malaria and filariasis.

It is therefore especially important that all possible measures be taken to effectively control their hazard to man, rather, than prohibiting their use in malaria eradication programs. Should the attitude of prohibition have been taken by the industrial hygienist, and in my opinion this is an industrial hygiene problem, many chemicals which have resulted in higher living standards could never have been produced.

Through an adequate safety program, dieldrin and other organic insecticides of comparable toxicity can be incorporated into malaria eradication programs with a minimum of hazard to the workers.

The elements of an effective safety program are:

1) Health education.
2) Good equipment and its proper maintenance.
3) Adequate and properly used protective clothing and equipment.
4) Proper labeling of containers.
5) Good personal hygiene.
6) Supervision, including disciplinary measures.
7) Medical supervision, including proper selection of workers and frequent medical examinations for evidence of preclinical poisoning.
8) Transfer of workers who evidence a sensitivity to organic insecticides.

SUMMARY AND CONCLUSIONS

In summary, I should like to quote the conclusions of the WHO Study Group on the Toxic Hazards of Pesticides to Man. These conclusions are most pertinent to the present discussion and summarize excellently the status of the insecticidal problem. «The increasing use of chemicals for insecticidal and similar purposes has not been matched by a corresponding increase in the effort spent on protective measures. More work is needed, particularly in the direction of providing complete field and toxicity data for the range of chemicals and methods of application in general use, so as to permit the formulation of safety regulations. These should be standardized on an international basis and should related the detailed method of application of chemicals (including such details as the maximum permissible concentration of the active ingredient and the amount sprayed per acre, etc.) to the protection required. A parallel development of suitable respirators (and clothing) will also be necessary».

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THE DEVELOPMENT OF OCCUPATIONAL HEALTH SERVICES IN LATIN AMERICA[1]

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In the long history of civilization, there has come a time in the existence of many nations when they have turned from an economy based solely on agriculture to one of diversified activities. That change has been accomplished with more or less consciousness and foresight in different areas.

In our times, with the amassed economic lessons of the industrial age widely available, certain less developed countries are deliberately working to foment all possible aspects of their economy — agriculture, manufacturing, mining and the exploration of other resources — and are calling on all available techniques to help them do so. They are trying to raise the technical skills among their people and to increase productive efficiency, so that they may develop industries of the mass production type. At the same time, they are using education to instill in people a desire for more and better goods and the wish to improve their way of living.

Farsighted leaders in these nations have put their influence into the enactment of progressive social legislation, bringing such benefits as health and maternity insurance, pensions for widows and orphans, old age and death pensions, and worker's compensation for accidents and occupational diseases. Industrial workers and their dependents have rightfully been favored in this trend toward social security, but

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[1] Paper to be presented at the Sixth International Congresses on Tropical Medicine and Malaria before Sub-section 2 (Housing and Industrial Hygiene) of Section 6, Tropical Hygiene and Sanitation, Lisbon, Portugal, September 5-13, 1958.

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these nations are learning the hard lesson that progressive legislation in itself can become a burden, without the preventive programs to reduce the disability both in and out of industry for which compensation is provided. Nations now developing their industries are, therefore, profiting by the experience of older industrial nations and are drawing on their techniques in occupational health as well as in general public health programs. One of the areas where there has been an unprecedented activity in the development of occupational health programs in recent years is that vast section of the Western Hemisphere known as Latin America.

Latin America encompasses the 20 American republics south of the United States. Its estimated population of 170 million people is scattered through regions varying in climate from temperate to tropical and in a few instances, such as in the high Andean plateaus and in the very southern sections of Chile and Argentina, cold winters are experienced. However, it has been estimated that approximately 40 percent of the population lives and works under what might well be considered tropical conditions and hence experiences many of the same health problems found in other tropical areas of the world. It should be of interest, therefore, to those concerned with health problems in tropical areas, to learn what has been accomplished in one such area in the development of health services in the interest of those gainfully employed. These services have been developed through the technical cooperation programs of the United States government.

The Institute of Inter-American Affairs, an agency of the United States Government, has been engaged in public health activities in Latin America for the past sixteen years, working in cooperation with the national governments. In each country, the two governments have carried out the joint program through an agency set up for the purpose. In some countries this agency is known as the Inter-American Cooperative Public Health Service.

These cooperative programs, which sprang out of resolution that health and sanitary measures were basic to mobilization activities at the beginning of the second world war, have transcended the war period, because it was recognized that these functions are equally vital in time of peace. Consequently, the governments of these countries and that of the United States of North America are carrying out health and sanitation programs in nearly all of the countries in Latin America.

Under the sponsorship of this program, hospitals health centers, water supplies, sewerage systems and other health and sanitary facilities have been constructed. Numerous programs have been undertaken in preventive medicine against major disease problems. In addition, many professional personnel from these countries has received training in public health in the United States and in other countries.

In the occupational health field, however, action did not begin until late in 1945, when the Bolivian government requested the United States to study the serious health problems among Bolivian workers, particularly those employed in the mining of tin.

I have been identified with the Institute's activities in the development of cooperative programs in the occupational health field, since its very beginnings. The programs which we have organized and developed have been based on a detailed study of the health problems in industry in each country. It is only after such a study of the problems of occupational health of a country and the resources available for dealing with them that it is possible to organize and develop an effective program. Studies of this character have been carried out in Bolivia, Brazil, Chile, Colombia, Ecuador, Mexico, Paraguay, Perú, Panama, Uruguay and Venezuela.

OCCUPATIONAL HEALTH PROBLEMS IN LATIN AMERICA

The studies that I have made in Latin America show in general that a serious obstacle in the development of the rich resources of these countries is the predominance of diseases which debilitate the worker and restrict the volume of production. Diseases such as diarrhea and enteritis and those caused by intestinal parasites are very common and serious. Respiratory diseases, such as pneumonia and tuberculosis, figure among the principal causes of death. Likewise, malaria is a serious problem in many of these countries.

When one studies the health problems directly connected with the working environment one encounters in these countries numerous hazards to health and to safety. Diseases such as silicosis, poisoning from toxic metals, gases, vapors and fumes are very common. Occupational health practices for the control of accidents and occupational diseases are practically unknown. In general, social welfare legislation shows considerable progress but unfortunately, this type of legislation has placed more emphasis on compensation than on prevention. The studies which I have made indicate clearly the high incidence of sickness and the excessive costs which industry pays in compensation.

As a matter of fact, in the Latin American countries where I have made studies, I have obtained sufficient data to show that these countries are spending large sums of money in compensation payments to workers for disability incurred on the job. These sums, in terms of national incomes and budgets represent approximately 15 percent of national incomes and from 30 to 50 percent of the national budgets.

The picture which I have just painted may at first sight appear to be very discouraging, but actually the future is very bright, since the lack of good occupational health programs can not be charged to any indiference on the part of the governments nor to industry. On the contrary, in a sincere effort to improve existing conditions which they know to be bad, several of the countries have asked the cooperation of the United States Government. In view of the existence of these conditions and of the desire to do something to remedy them, this is the precise time to develop practical and effective programs in occupational health in many of the Latin American countries, especially in those that are rapidly industrializing. Considering that these countries have as yet a great deal to accomplish in the general field of public health, such programs are especially appropriate for them. A strong and vigorous occupational health program can serve as a medium to stimulate the practice of general public health.

RECENT DEVELOPMENTS IN OCCUPATIONAL HEALTH IN LATIN AMERICA

Many Latin America countries, in an urge to improve the living standards of their workers, have enacted social legislation which can be considered among the most progressive in the world. However, much of this legislation, in spite of being well conceived, has been only partially implemented because of a lack of administrative machinery. These countries have had many years of experience with legislation concerned with compulsory sickness insurance, old age benefits, benefits in case of death, indemnities to workers for accidents and occupational diseases, and similar laws designed to assist the worker if the necessity presents itself. It is evident that the predominant point of view has been compensation, that is, assisting the worker after the damage has been done.

It cannot be denied, however, that some effort has been made to establish programs of occupational health in many of these countries. Practically each country in Latin America has its division of Occupational Health, located either within a Ministry of Public Health or a Ministry of Labor. Unfortunately, in the past, the majority of these division did not have trained personnel, nor facilities to carry out a truly scientific program of prevention of occupational health hazards. Even today, industry in many of these countries does not have available the services of specialists who are trained to study occupational health problems and to advise on how they may be solved. Without modern methods for the study of these problems, the occupational health division of Latin America, up to recent years, have attempted to evaluate these problems, using solely their senses of sight and smell, which, as all of you know, are totally inadequate and deceiving. Moreover, the few programs that were carried out under the same of occupational health had been limited principally to the study and control of occupational diseases, especially such diseases as silicosis and lead poisoning.

The most important development in occupational health during the last twenty years, especially in the Western Hemisphere, has been a change in emphasis from the mere control of occupational diseases to the improvement of the general health of the workers and their families. This is precisely the reason why in many countries occupational health services have been organized as an integral part of public health agencies. This change in the objective of the program is because it is well known that it is not sufficient to maintain the worker free from occupational diseases alone and that any health program which merits the name has to go beyond the occupation of the man and consider him as a member of a community. As a matter of fact, if we should concern ourselves solely with occupational health problems, we would be limiting our efforts to a mere ten percent of the health problems in industry and overlook the principal causes of disability, which are of a non-occupational character. The necessity to include these non-occupational disabilities within our occupational health programs in Latin America is very evident from the health picture just discussed.

It is obvious then, that today the objectives of occupational health carry out health programs toward three goals: 1) a safe and healthful working environment; 2) a healthy community environment in which the worker lives; 3) healthy and well-adjusted men and women engaged in industrial production.

In considering these three goals, we must not forget the principal objective of industry: to obtain maximum production of goods and services in the most efficient and economic manner. Occupational health has to be completely realistic.

Now, let us considerer the recent developments of occupational health in Latin America.

According to the estimates based on the last census in Latin America there are approximately 170 million inhabitants living in the twenty republics and out of this number some 60 million are gainfully employed. During the last thirteen years, the Institute of Inter-American Affairs has given assistance in the development of occupational health programs to twelve countries which include about 80 percent of the labor force of Latin America.

The philosophy employed in the development of these occupational health programs is very similar to that used by the United States Public Health Service to carry out its work among the States in that country. There, the principal objective was the organization within a State Public Health Agency of a group of trained personnel backed up by the essential laboratory and field equipment, so that occupational health services could be brought to the industries of the area under consideration.

Such a plan can be achieved with some success in a country where the public health practice is well established along modern standards. Lacking a similar public health structure in some of the countries where we are working we have found it more convenient in some of them to administer the occupational health programs during their first years of existence under the guidance of United States personnel. In this manner, we have developed modern occupational health programs in most of the Latin American nations, except in a few predominantly agricultural countries. In all of these countries we employ United States consultants who cooperate in the development of these program in their initial years. All of these programs have been organized and developed under special cooperative project agreements as part of the general cooperative agreement between the Institute of Inter-American Affairs and the host country.

It is our ambition to develop within each of these countries where we are working, occupational health programs whose principal functions will be to guide and advise industry in the solution of its health problems and to stimulate large industries to establish their own occupational health activities. As the countries gain experience in the administration of their programs we gradually turn them over to them so that they themselves may operate them. Also we hope to continue sowing the seeds of occupational health knowledge through the presentation of lectures in the various professional schools, such as those now being given in Chile, Brazil, Mexico and Venezuela. Likewise, we plan to continue our training of outstanding personnel by sending them to the United States and elsewhere to study.

We are urging the various countries that they enter into the program of health education of their workers through the means of educational films and literature, especially written for them. We are also stimulating the various countries to organize occupational health associations made up of members from government, industry, labor and the professions, for the purpose of serving as a vital force which can unite all of the resources of industry, of labor, and of government in a cooperative program for the protection and improvement of the health of the millions of workers in Latin America.

REMAINING PROBLEMS

From the above description it is evident that occupational health is making progress in Latin America. However, there are still many problems to be resolved, a few of which result from the very political and social philosophy of these countries, which has prevailed for centuries as a way of life of these nations. For example, in many countries government workers are very badly paid, forcing them to have several jobs to which they can only devote a few hours a day to each one. In those countries which require a full day's work in which the salaries are low, there is a constant turnover of personnel. Such a state of affairs is not conducive to professional stability, nor to continuity in the programs. The countries in which such conditions prevail will have to recognize very quickly that in order to maintain a loyal and well-trained professional staff, they will have to take steps to assure that their employees are paid in accordance with their efforts and that they have tenure in their jobs.

In several countries where we are working, the teaching resources in the field of medicine, engineering and chemistry are somewhat inadequate, which makes it difficult to recruit suitable professional personnel for work in occupational health. The Institute of Inter-American Affairs is trying to remedy this situation by means of contracts between United States universities and those in Latin America, for the purpose of strengthening the teaching methods in the latter universities. This is done through the interchange of professors, the granting of fellowships to professors for study in the United States, and the training of Latin American professionals in the country of origin. To date, several projects of this kind have yielded satisfactory results.

One of the best-intentioned but most deceiving laws that I have encountered in my work is the so-called law of unhealthy occupations — a law which exists in quite a few Latin American countries. For instance, according to such a law, a worker through the intervention of his union may request the government agency responsible for administering the law to determine if his job is unhealthy or not. If, after investigation of the working conditions, the job is declared unhealthy, the worker either receives more pay or he need only put in six hours of work a day but will receive pay for eight hours. It is obvious that such a law does nothing to change the working environment and that it does not favor any one in spite of its good intentions. Actually, everybody stands to lose through such a law. Industry is obliged to pay for work it does not receive and the worker with his extra money can never buy a new pair of lungs or kidneys, if these should happen to be damaged because of his work. In actual practice, the only interest that the worker has is to obtain more money instead of the true objective, which is to control the bad working conditions.

Moreover, in some countries, where the law exists, the government agency in charge of its administration does not have the qualified personnel, nor the field or laboratory equipment necessary to determine if a job is truly dangerous or not. It would be much better, if the governments where these laws exist would withdraw them and in their place provide for the organization of a modern occupational health program, one provided with trained professional personnel and the essential field and laboratory equipment necessary to study and remedy the dangerous conditions which exist.

This brief sketch on the growth of occupational health in Latin America, demonstrate that in the last thirteen years the governments of the countries in that area have only taken the first steps to establish such programs on a solid and modern base. Even so, they can well serve as an example to other nations, be they in tropical areas or not, which are desirous in developing similar programs. Fortunately, the majority of the problems with which industry in these nations is confronted today are of the type well-known and for which solutions are available. The health hazards which modern industry is faced with in the more industrialized nations, such as in the United States and in the nations of Western Europe, are only now beginning to appear in Latin America. When the latest technological developments become a part of the life of these countries, Latin America should be ready to solve newer problems through its modern occupational health programs, programs which meanwhile are contributing to the health, welfare ad productivity of the millions of workers who live in the southern part of the Western Hemisphere.

GENERAL DISCUSSION

Dr. G. P. Crowden (U. K.) — Referring to the problems of protective clothing raised by Dr. Macpherson and Dr. Doges — drew attention to the value of wearing a wet cotton jacket outside the non-ventile protective clothing.

Dr. Chatterjee who had followed up earlier research on gas protective clothing with experiments on industrial protective clothing had shown this measure to be effective in cooling the worker in hot environments. Also that air supplied to the helmet from an electric air pump used for inflating motor tyres not only relieved respiratory distress but also helped vision by preventing the misting up of the visor of the protective helmet.

Referring to radiant heat stress in industry Dr. Crowden quoted a case in which this was prevented by painting the hot surface of plant with aluminium paint of 70 % emmissivity, and interposing a heat reflecting sheet of aluminium between the hot plant and the worker. This measure not only benefitted the worker but also it was found that less electricity was required to heat the plant as less heat was given out by radiation.

SUBSECTION 3

SANITARY ENGINEERING

1ST SESSION: *Wednesday, September, 10th — 11:00 to 12:30 a.m. Room C*

The meeting was held under the Chairmanship of Dr. John Bloomfield.

The Rapporteur was Eng. Arturo Luis Berti and the Secretary was Eng. Salomão Q. Levy.

The following papers were presented and discussed:

IMPORTANCIA DEL CONTROL DE MOSCAS EN LA INCIDENCIA DE DIARREAS Y ENTERITIS EN LOS TROPICOS

ARTURO LUIS BERTI [1]

Jefe, División de Malariologia Maracay, Aragua, Venezuela

Especialmente después del adelanto de las campañas antimaláricas, realizadas en estos últimos años en los trópicos americanos, las diarreas y enteritis han venido ocupando uno de los puestos más importantes entre los problemas que afectan la salud de los habitantes. Posiblemente en otras regiones de la zona tropical se haya observado también este fenómeno. En los países de la Región Neotrópica, las diarreas y enteritis constituyen en la actualidad una de las tres principales causas de muerte. En 10 de esos países tales tasas están por encima de 100 por 100 000 habitantes, alcanzando en dos de ellos, El Salvador y Guatemala, cifras verdaderamente alarmantes: 342 y 357 en 1952. Por otra parte, el grado de ruralidad tiene gran influencia en la prevalencia de estas enfermedades. En Venezuela la más alta mortalidad ocurre en localidades de menos de 1 000 habitantes (Curiel y de Ochoa, 1957), justamente en donde es más difícil el desarrollo de la asistencia médica adecuada y de las más convenientes medidas de saneamiento. Por este motivo se impone la búsqueda de métodos de control que puedan ser económica y eficientemente aplicables a las zonas rurales para conseguir dentro de un tiempo razonablemente corto progresos efectivos en este campo

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[1] De la División de Malariología, Dirección de Salud Pública, Ministerio de Sanidad y Asistencia Social, Maracay, Aragua, Venezuela.

Las nuevas técnicas de medicina curativa empleadas en el campo de las gastroenteritis y colitis disminuyen la letalidad grandemente. Se basan en la educación del médico para que éste sea capaz de ejecutar una asistencia edecuada a cada caso. Sin embargo, el problema en la comunidad permanece intacto, pues ninguna repercusión tiene el tratamiento sintomático de algunos enfermos en la reducción de la transmisión, de lo cual depende en última instancia la diminución de la morbidad y consiguientemente de la mortalidad. Por otra parte, es justamente en las zonas rurales en donde el número de médicos es más escaso, problema que no parece tener pronta solución en las zonas tropicales.

En cuanto a las medidas de saneamiento utilizables, sabemos que ellas pueden estar dirigidas a atender: *a*) las fuentes de infección, *b*) los factores de transmisión, y *c*) los agentes que faciliten la infección o la transmisión.

En cuanto al saneamiento de las fuentes de infección, deberá atenderse el problema constituido por las excretas humanas, los animales domésticos y otros animales. Una mejora rápida en disposición de excretas es cosa difícil en las zonas rurales por motivos económicos y educacionales. Tampoco es posible en dichas zonas eliminar facilmente la influencia de los animales domésticos y de otros animales tales como ratas y murciélagos.

Con referencia al saneamiento de los factores de transmisión es de todos conocida la dificultad de obtener en las zonas rurales un suministro de agua adecuado en el interior de cada casa, lo cual es necesario pues no sólo es el agua que se bebe la que debe ser pura sino que también se necesita agua en abundancia para la limpieza, factor este último que tiene con frecuencia más importancia que el primero en quanto al uso del agua se refiere. También es poco lo que puede hacerse en la higienización de los alimentos, salvo la cocción de la leche que es medida amplamente usada en los trópicos americanos y que debe ejercer un eficiente papel profiláctico. El papel de los fomites está en razón inversa del grado educacional de los habitantes, lo cual es por lo común bajo en las zonas tropicales y difícil de mejorar en corto tiempo. La suciedad de la manos es también asunto de dificultosa corrección, puesto que en gran parte depende del suministro de agua adecuado. El control de moscas, como consecuencia de las recientes adquisiciones en el ramo de los insecticidas, es un campo a explorar.

Con respecto al saneamiento de los agentes que facilitan la infección o la transmisión se sabe que la vivienda juega un papel importante, pero el mejoramiento de la vivienda no es empresa que puede llevarse a cabo en corto tiempo. Igualmente la suciedad de los arrabales en el medio urbano constituye problema de difícil solución. El mejoramiento del medio rural está supeditado al progreso aducacional y económico de la población, asunto cuya solución no se vislumbra en un futuro próximo.

Dadas las dificultades anteriormente enumeradas, vale la pena explorar la relación que las moscas tienen como factores de transmisión de las diarreas y enteritis, y si se encuentra que ellas son responsables de una proporción importante de la transmisión, ver los resultados que pueden obtenerse con el uso de los modernos insecticidas. Es a este punto al que se limita la presente exposición.

MOSCAS Y DIARREAS Y ENTERITIS

A pesar de que por más de medio siglo se supo que las moscas podían transmitir las bacterias productoras de diarreas y enteritis, no fué sino hasta la introducción de los insecticidas de acción remanente que se vino a valorar la importancia que tales insectos tenían como vectores de dichas enfermedades. Observaciones llevadas a cabo en la Zona Templada (Missiroli, 1948; Watt y Lindsay, 1948) y en la Zona Tropical (Gabaldón, 1949; Berti, 1949) pusieron de manifiesto que las moscas eran elemento de gran importancia en la transmisión de las enfermedades diarréicas.

Varias son las especies de moscas que se han encontrado en la naturaleza infectadas con bacterias productoras de enfermedades diarréicas. Sin embargo, la importancia de una especie está en relación directa con su domesticidad. En Venezuela las moscas más frecuentemente encontradas en el ambiente doméstico o peridoméstico son: *Musca domestica, Sarcophagula* spp. y *Drosophila* spp. La primera es conocida desde hace mucho tiempo como vector de estas enfermedades pero el papel transmisor de las dos últimas ha sido determinado recientemente por el Dr. Eugenio Angelillo en los Laboratorios de la División de Malariología. El estudio de la Figura 1 señala con gran claridad que en Venezuela la distribución estacional de la densidad de moscas está en íntima relación con la distribución estacional de la mortalidad por diarreas y enteritis. Este hecho también ha sido encontrado en otros países, especialmente en la Zona Templada, en donde hoy se considera que las diarreas de verano son debidas mayormente al aumento de la transmisión por el incremento de la densidad de las moscas en dicha estación.

Queda por saber qué proporción de los casos de diarreas y enteritis que se presentan en los trópicos son debidos a transmisión por moscas. Esto dependerá naturalmente del *dintel de transmisión,* es decir, la densidad numérica por debajo de la cual las moscas no pueden mantener la transmisión en la comunidad, lo cual debe ser variable de acuerdo con el grado de saneamiento ambiental que ésta posea. Por ejemplo, en una ciudad en donde todas las casas estén dotadas de excusados de agua y en donde no haya animales domésticos que rieguen heces en los terrenos inmediatos, el *dintel de transmisión* podrá ser más alto que en otra ciudad en donde la disposición de excretas sea en el suelo o en letrinas. Por este motivo es de esperar que una reducción numérica de moscas tenga un papel diferente en distintos lugares, y que en todo caso la efectividad de las medidas contra las moscas será ayudada o dificultada por la presencia o ausencia de buenos sistemas de disposición de excretas.

El papel primordial jugado por las moscas en la transmisión de enfermedades diarréicas lo revelan los estudios de Weir (1952) realizados en aldeas egipcias, los cuales demuestran que aparentemente todo el peso de la transmisión estaba a cargo de esos insectos. En Venezuela, en donde la densidad de moscas es menor que en Egipto, alrededor del 50 por ciento de los casos parece ser debido a transmisión por moscas (Gabaldón, 1955; Gabaldón, Berti y Jove, 1957). En efecto, nuestros estudios demostraron que el número de casos de diarreas y enteritis asistentes a un dispensario médico de una aldea rebajó a la mitad después de la introducción del rociamiento de dos gramos de DDT por metro cuadrado en 1946, y se recuperó en 1949 cuando apareció resistencia de las moscas al insecticida. También indican nuestros estudios que en el área no malárica de Venezuela, en municipios con un médico por cada 5 800 habitantes, las tasas de mortalidad por diarreas y enteritis en el período 1946-1954 permanecieron sin alterarse entre 176 y 187 por 100 000 habitantes. En otro grupo de municipios, con un médico por cada 10 843 habitantes, cuyas casas fueron rociadas para el control de triatominos, la tasa de mortalidad por diarreas y enteritis, que fué en el período de 1946 a 1950 de 205, bajó a 91 en 1954.

Ante los hechos que se acaban des enumerar es evidente que el control de moscas constituye una de las acciones sanitarias fundamentales a realizar para obtener la reducción de las diarreas y enteritis en un tiempo razonablemente corto. Este hecho deberá significar un cambio apreciable en la orientación de las labores sanitarias en las zonas tropicales, puesto que ùltimamente ha estado muy descuidada toda acción contra las moscas, y con frecuencia enteramente olvidada por los servicios sanitarios.

CONTROL DE MOSCAS

Las medidas de saneamiento dirigidas a disminuir la población de las moscas pueden englobarse en los dos grupos siguientes: *a*) medidas antilarvarias encaminadas a la eliminación de los criaderos más que a la matanza de larvas; y *b*) medidas anti-imagales, que tienen por objectivo la muerte de los adultos.

Para la aplicación de las medidas antilarvarias se requiere de un estudio previo con el fin de determinar cuales son los criaderos de mayor significación. En Venezuela se ha encontrado diferencia entre la importancia de determinados tipos de criaderos en el medio urbano y en el medio rural. Lo mismo debe suceder en otros países de la Zona Tropical.

En el medio urbano existen grandes focos de producción y pequeños focos de producción larvaria. Esto no quiere decir que los primeros sean más importantes que los segundos, pues con frecuencia los segundos son muy numerosos y esto basta para que sean responsables de altas densidades de moscas. Entre los primeros se encuentran los mercados y los mataderos y con menor frecuencia los establos para caballos, vacas y cerdos, en los alrededores de las ciudades. Los trabajos de control de moscas en mercados y mataderos consisten en labores generales de limpieza para la eliminación de los residuos, que en nada difieren de lo aconsejado en cuanto a almacenamiento, disposición y recolección de basuras. En los establos son los excrementos de animales los criaderos principales y no es siempre fácil ni económico hacer lucha antilarvaria en ellos, pues la recolección y disposición de tales excrementos significa una labor costosa.

Los pequeños focos de producción larvaria en el medio urbano están constituídos en Venezuela principalmente por los desperdicios domésticos, y en menor cuantía, según el grado de saneamiento existente, por excrementos animales e humanos. Por ejemplo, en 605 casas de Maracay, estudios practicados por el Dr. Corrado Bianchini en 1953, demostraron em 576 criaderos la distribución siguiente:

	Desperdicios domésticos	Excretas animales	Excretas humanas	Total
Musca	334	6	57	397
Sarcophagula	41	23	77	141
Drosophila	26	5	16	47

Los datos anteriores revelan la necesidad de perfeccionar el servicio de recolección y disposición de basuras en las ciudades como base fundamental para la eliminación de esos criaderos dentro de las casas. Tal acción tendrá en Venezuela un efecto mayor en el control de *Musca domestica* puesto que son los desperdicios citados los que constituyen dentro de las casas los criaderos más frecuentes. Esta suposición la confirman los siguientes datos de Maracay, ciudad en donde en febrero de 1956 se reorganizó el Servicio de Aseo Urbano y Domiciliario. Las cifras que en seguida se exponen son los índices de densidad de moscas por casa (Berti y Gómez, 1957) y corresponden al mes de mayo cuando *M. domestica* alcanza su mayor densidad:

Años	*Musca*	*Sarcophagula*
1953	44,8	4,2
1954	33,0	4,0
1955	32,6	9,3
1956	14,2	6,6
1957	15,8	6,7
1958	11,1	6,6

Se vé con claridad que *Sarcophagula*, representada aquí por las especies *S. canuta* y *S. occidua*, cuyos criaderos principalmente son los excretos humanos y animales, no sufren alteración en cuanto a su densidad como consecuencia de la mejora en la recolección y disposición de las basuras. Por el contrario *M. domestica* si ha tenido una franca disminución, hecho que era esperado por cuanto los desperdicios domésticos constituyen sus principales criaderos y son justamente los eliminados por el Servicio de Aseo Urbano y Domiciliario.

Como se demostró anteriormente, los excretos humanos constituyen criaderos importantes de moscas. Con el fin de demostrar si las letrinas, es decir, los excusados de hoyo, son factor de producción de moscas, el Dr. Corrado Bianchini realizó 1 261 observaciones en 217 letrinas cerca de Maracay y encontró la siguiente producción de moscas por observación en cada 24 horas:

Profundidad (cm)	*Musca*	*Sarcophagula*	*Drosophila*
0-100	15,6	2,5	44,9
101-200	5,0	1,2	47,9
201-300	6,8	0,9	67,2
+ 300	0,8	0,5	57,9

Las cifras anteriores demuestran que *M. domestica* se cria en letrinas superficiales y que es prácticamente eliminada en las que tienen más de tres metros de profundidad. Por otra parte, las especies del género *Drosophila*, representadas especialmente por *D. ananassae*, tienen aparentemente una producción mayor cuanto más profunda sea la letrina. Esto indica que las letrinas, si bien constituyen una medida de saneamiento eficiente en el control de helmintiasis, particularmente de la anquilostomiasis, son por el contrario un peligro en cuanto a la transmisión de las diarreas y enteritis. Recuérdese que *D. ananassae* ha sido hallado en Venezuela infectada con bacterias del género *Shigella* y que ésta es una mosca que entra mucho en las cocinas y reposa sobre frutas que se comen frescas. Como los insecticidas aplicados en las letrinas aumentan la producción de moscas producidas en ellas (Kilpatrick y Bogue, 1956) se hace difícil el control a menos que se emplee protección mecánica por telas metálicas.

Ahora bien, las labores de recolección y disposición de basuras no pueden llevarse a cabo sino en ciudades que puedan sostenerlas. Pero estos trabajos no eliminan todos los criaderos existentes y conviene por ello hacer un trabajo adicional en los grandes focos de producción: mataderos, establos, etc. Sin embargo, en estos medios dan más eficiencia las medidas anti-imagales que las antilarvarias. El sistema de cuerdas impregnadas con insecticidas fosforados es posiblemente la medida anti-imagal más fácil de realizar, pero aunque costosa es más barata que otros métodos. La eficiencia de esta medida lo revela las cifras presentadas en el Cuadro nº 1 en el cual se ve que durante cuatro años se ha mantenido muy baja la densidad de moscas en un establo de vacas de 1 900 metros cuadrados bajo techo con 285 animales. Interesa observar en dicho cuadro que los índices han ido aumentando paulatinamente y aunque todavía están bajos es probable que indiquen el inicio de la aparición de una muy débil resistencia al *Diazinón*.

El método de cuerdas impregnadas, o medidas anti-imagales similares podrán ser los instrumentos de control de moscas en el medio rural. Necesítase sin embargo hacer estudios sobre sus costos y las dificultades organizativas y administrativas que puedan presentarse.

QUADRO 1 — *Control de moscas en un establo de vacas cerca de Maracay, Araguas, Venezuela, por medio de cuerdas impregnadas con Diazinón en suspensión al 5 por ciento*

Periodo	Impregnaciones de las cuerdas	Indice de moscas (medianas para el periodo)
Mayo, 1954	0	63,6
Julio-Septiembre, 1954	4	7,6
Octubre-Diciembre, 1954	4	4,5
Enero-Marzo, 1955	3	2,2
Abril-Junio, 1955	4	2,4
Julio-Septiembre, 1955	4	2,7
Octubre-Diciembre, 1955	4	4,6
Enero-Marzo, 1956	3	4,4
Abril-Junio, 1956	4	5,8
Julio-Septiembre, 1956	4	4,1
Octubre-Diciembre, 1956	4	—
Enero-Marzo, 1957	4	5,5
Abril-Junio, 1957	3	6,4
Julio-Septiembre, 1957	4	5,8
Octubre-Diciembre, 1957	4	5,5
Enero-Marzo, 1958	4	5,6
Abril-Junio, 1958	4	7,0

CONTROL DE MOSCAS E INCIDENCIAS DE DIARREAS Y ENTERITIS

La campaña contra la malaria en escala nacional por medio del rociamiento con DDT de las casas del área malárica comenzada en Venezuela en 1945 demostró ya para 1948 (Gabaldón, 1949; Berti, 1949) que la mortalidad general de las áreas protegidas con el insecticida había disminuido a cifras inferiores a las que se esperaba como consecuencia del puro decrecimiento de la malaria. Las enfermedades que más se había reducido eran las diarreas y enteritis, fenómeno que debió ser originado por el efecto que al principio tuvo el insecticida sobre la población de moscas, cuya densidad alcanzó cifras mínimas en los primeros años de las labores.

En Venezuela los municipios con servicio sanitario bien organizado, aquí llamados Unidades Sanitarias, disponen de relativamente buenas estadísticas de mortalidad, en las cuales se puede tener confianza. Un estudio hecho para investigar la diferencia de reducción de las diarreas y enteritis en municipios con Unidad Sanitaria, protegidos y no protegidos (Gabaldón, 1955) demostró que las tasas de mortalidad por diarreas y enteritis por 100 000 habitantes sufrieron los cambios siguientes:

	1941–1945	1950
Area no malárica:		
Municipios con Unidad Sanitaria, no protegidos con DDT.	274,4	254,4
Area malárica:		
Municipios con Unidad Sanitaria, protegidos con DDT	271,3	146,4

Estas cifras revelan que en los primeros cinco años del rociamiento, cuando todavía el insecticida tenía influencia sobre las moscas la tasa de mortalidad por diarreas y enteritis cayó casi a la mitad en los municipios protegidos, mientras que prácticamente no sufrió modificación alguna en aquellos municipios que no recibieron el insecticida. Estos datos son muy demostrativos, pues indican claramente que el control de moscas produce una rebaja notable en la incidencia de diarreas y enteritis, rebaja que se logra en corto tiempo. No importa que el insecticida empleado haya producido resistencia de las moscas, fenómeno que puede anular las vantajes alcanzadas, lo que importa es haber demostrado que una reducción en la densidad de la población de moscas se acompañó con un decrecimiento marcado de las tasas de mortalidad por diarreas y enteritis. Esto significa que si se desea conseguir resultados semejantes habrá que valerse de nuevos insecticidas o de otros procedimientos dirigidos a disminuir las moscas como punto fundamental en el control de las diarrreas y enteritis en nuestro medio.

Los estudios antes citados también señalaron que la disminución de la mortalidad por diarreas y enteritis fué más intensa a medida que la proporción de casas protegidas era mayor. Los municipios del área malária recibieron de 1945 a 1950 distintos grados de protección de acuerdo con la prevalecencia de la malaria, pues había unos municipios en donde dicha enfermedad estaba presente solo en una pequeña porción de su territorio y otros en donde todo el territorio estaba afectado. Se dividieron los municipios en cuatro grupos y como lo demuestran las cifras siguientes, aquellos que recibieron mayor protección fueron los que en 1950 tenían más bajas las tasas de mortalidad por diarreas y enteritis.

	1941-1945	1950
Municipios protegidos menos del 25 %	239,8	188,1
Municipios protegidos del 25 al 49 %	299,3	141,0
Municipios protegidos del 50 al 74 %	266,0	135,6
Municipios protegidos más del 75 %	207,0	116,1

Es evidente que mientras mayor fué la proporción de la población de moscas afectada por el insecticida, mayor fué la reducción alcanzada en la mortalidad por diarreas y enteritis. Esto viene pues a confirmar la posibilidad de llevar a cabo en un tiempo relativamente corto un cambio radical en la incidencia de diarreas y enteritis al introducir un control adecuado de moscas.

En la ciudad que tiene mayor número de años de reorganizado su Servicio de Aseo Urbano y Domiciliario, según pautas de la División de Malariología y bajo el control del personal de ésta, es decir, Maracaibo, se ha alcanzado también un efecto benéfico en la reducción de diarreas y enteritis. Ya anteriormente citamos las cifras de cómo disminuyeron las moscas en esta ciudad, y véase enseguida la rapidéz conque cayeron las tasas de mortalidad por diarreas y enteritis en dicha ciudad a partir de 1951, el año anterior a la reorganización del Servicio.

Años	Tasas de mortalidad
1951	228,7
1952	186,8
1953	129,8
1954	124,1
1955	84,3
1956	106 3
1957	109,9

Las cifras anteriores demuestran la importancia que tiene en las ciudades tropicales una buena organización del Servicio de Aseo Urbano y Domiciliario que produzca una adecuada reducción de moscas. Esta reorganización debe ser acompañada de servicios antilarvarios complementarios en los grandes focos de producción, asunto este que no se ha podido todavía llevar a cabo en las ciudades en donde hemos trabajado.

Aunque no disponemos de cifras sobre densidad de moscas sí se sabe que en otra ciudad, Trujillo, hubo desde 1953 un inconveniente en la disposición de las basuras que produjo un aumento de la densidad de moscas, lo cual repercutió en un aumento de las tasas de mortalidad por diarreas y enteritis como se vé en las cifras siguientes:

Años	Tasas de mortalidad
1948	233,4
1949	166,8
1950	219,2
1951	127,7
1952	258,7
1953	474,7
1954	521,3
1955	610.1

La gran influencia de la mortalidad por diarreas y enteritis sobre la mortalidad infantil la prueban también las cifras de Trujillo. En 1951 hubo la tasa más baja de mortalidad por diarreas y enteritis: 127,7, la cual se acompañó de una tasa de mortalidad infantil de 71,7, que fué también la más baja en todo el período de 1946 a 1955. Por el contrario, cuando en 1955 la tasa de mortalidad por diarreas y enteritis se elevó a 610,1, la tasa de mortalidad infantil aumentó a 165,8 y esas tasas fueron también las más altas observadas en el mismo período de tiempo. Se ve entonces cómo el incremento en la densidad de moscas a que se hizo referencia tuvo tan profundas repercusiones en los dos índices nombrados. Es de advertir que en todo ese tiempo las medidas de higiene materno-infantil practicadas en la ciudad fueron de la misma calidad. Este es un nuevo ejemplo de la importancia de mantener un control adecuado de las moscas si se pretende conservar unos índices sanitarios pasablemente buenos. También indican con gran claridad estos hechos que la efectividad de las medidas de medicina preventiva ejecutadas en una localidad dada, es en gran parte anulada por insuficiencias en el saneamiento ambiental.

SUMARIO

A pesar de que por más de medio siglo se supo que las moscas podían transmitir las bacterias productoras de diarreas y enteritis, no fué sino hasta la introducción de los insecticidas de acción remanente que se vino a valorar la importancia que tales insectos tenían como vectores de dichas enfermedades. Varias son las especies que se han encontrado en la naturaleza infectadas con bacterias productoras de enfermedades diarréicas. Sin embargo, la importancia de una especie está en relación directa con su domesticidad. En Venezuela las moscas más frecuentemente encontradas en el ambiente doméstico o peridoméstico son: *Musca domestica*, *Sarcophagula* spp. y *Drosophila* spp.

El papel primordial jugado por las moscas en la transmisión de enfermedades diarréicas lo revelan los estudios de Weir (1952) realizados en aldeas egipcias, los cuales demuestran que aparentemente todo el peso de la transmisión estaba a cargo de esos insectos. En Venezuela, en donde la densidad de moscas es menor que en Egipto, alrededor del 50 por ciento de los casos parece ser debido a transmisión por moscas. Ante los hechos que se acaban de enumerar es evidente que el control de moscas constituye una de las acciones sanitarias fundamentales a realizar para obtener la reducción de las diarreas y enteritis en un tiempo razonablemente corto. Este hecho deberá significar un cambio apreciable en la orientación de las

labores sanitarias en las zonas tropicales, puesto que últimamente ha estado muy descuidada toda acción contra las moscas, y con frecuencia enteramente olvidada por los servicios sanitarios.

En Venezuela la principal acción en cuanto a medidas antilarvarias ha sido la reorganización de los Servicios de Aseo Urbano y Domiciliario en las ciudades. Para grandes focos de producción, como establos y mataderos, se han empleado medidas anti-imagales a base de insecticidas fosforados. Ambos procedimientos se han acompañado de una disminución considerable de la densidad de moscas. Esto ha significado, como se demuestra en varios ejemplos, una reducción de la mortalidad por diarreas y enteritis. Parece ser que el control de moscas constituya en los trópicos la medida más rápida de influencia en la mortalidad por estas enfermedades.

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SANITARY COLLECTION AND DISPOSAL OF ORGANIC WASTES

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Arrangements of the collection of organic wastes barely existed in primitive villages and medieval towns yet the level of health enjoyed by members of these communities was not seriously affected by even the total absence of sanitary arrangements. Some ancient civilisations it is true, were conscious of the dangers to health and comfort which insanitary conditions in close living communities carry, but man is not an inherently fastidious creature. So long as he has one foot in the countryside with plenty of clean air with easy access to fresh food and pure water, the consequential diseases of insanitary conditions are restrained by natural cleansing and protective agencies and then he appears to prefer to tolerate these diseases in their limited manifestations rather than apply himself diligently to remove the cause. It was not indeed, until «dirt» diseases caused death on a catastrophic scale within rapidly expanding towns and industrial cities during last century that it was realised that efficient sanitary services were essential provisions if man was to live in comfort and sound health within urban communities. Thus within the short space of one hundred years we have seen sanitary services developed to reach a high pitch of efficiency and it is within the great cities of the world, once so insanitary, that we find man living in the cleanest of all environments.

Intricate and massive sewerage systems remove faecal matter and specially designed receptacles and vehicles make it possible for other organic wastes to be removed without offending against the

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[1] The author did not attend the meeting.

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comfort or health of the town dweller, and a distinction is now made between putrescible organic wastes which alone offend and inert wastes which are merely an inconvenience. The separate collection of dry organic wastes, particularly kitchen wastes, which is commonly practised in many large towns in the West, was largely dictated by the exigencies of war. This material was required to contribute to food production. During wars, peace time standards in many aspects of life have to be lowered and extra demands freely accepted which would otherwise not be tolerated. Thus the communal bin and the extra trouble it involved into which scraps of food from surrounding houses were placed was freely accepted by the busy housewife as was the nuisance of decaying matter of all kinds spilled from these bins by prowling dogs and cats. Now with a return to more normal conditions the communal bin has gone and those communities which are sufficiently prudent to recognise the advantages of conservation over destruction have substituted separate services for the collection of organic and inorganic dry wastes. The organic wastes thus collected being processed and sterilised and made suitable as animal feeding stuff.

In direct contrast to services providing for the collection of edible organic wastes in its dry state, the purpose of which is primarily one of conservation, the disposal of these wastes along with sewage is essentially one of convenience. The justification of the latter system being that since organic wastes are potentially harmful they should be removed from dwellings as quickly as possible. Grinding machines are provided which allows bulky organic matter to be reduced in size sufficiently to allow it to enter sewers and be transported and disposed of along with normal domestic sewage. The larger bulk of organic wastes in the sewers increases the water required to effect proper conveyance, by about ten per cent and adds to the problem of sludge disposal when the sewage is treated.

The rigid application of methods of organic waste collection, in dry or liquid form, which have been established in recent years however, makes it possible for this potentially harmful matter being removed hygienically and they can be applied equally well to the collection of night soil from simple latrines and privies. The humblest village can be as clean as the largest city. Indeed so effective can these methods be when properly applied that they can subject man to new dangers. Complete sterilisation denies him the protection that natural immunisation affords and if the full advantages of hygienic collection are to be enjoyed, disposal arrangements must be equally effective.

The natural function of waste organic matter is to improve or maintain the fertility of soils. When this matter is spread on the surface of the agricultural land and mixed with the topsoil, it decomposes to form humus. Fertile soil is a natural cleansing agency and the processes which result in the inoffensive decay of waste matter when in natural contact with the soil are biologically distinct from the parallel processes of putrefaction which come about if such matter is allowed to accumulate under unnatural conditions. For centuries past this has been known but the trend in sanitary practice during the whole of the past century has been to depart from the use of agricultural land as a medium in effecting the hygienic disposal of organic wastes. The result is that wherever man congregates in any numbers throughout the world the waters around him are polluted and the land scarred with dumps of putrefying wastes.

The use of human faeces for manuring has persisted long after the use of the variety of dry organic wastes stopped. Although the dangers attached to the use of human faeces for manuring have been generally exaggerated, the wholesale and sometimes indiscriminate use that has been made of it where primitive agricultural methods are practised has, while maintaining food supplies, caused widespread intestinal disorders of a variety of kinds. Simple precautions would eradicate this risk but as civilisation extends the prejudice against the use of faeces in the production of food for human diet increases and the rapid increase in the production of artificial chemical fertilisers does nothing to remove that growing prejudice.

The evidence against the use of faeces or sewage sludge in its crude state with simple precautions, as a manure is in fact, slender and unconvincing. When it is brought into contact with the soil it quickly loses any of the injurious properties it may carry. In its crude state however, it is fundamentally an nitrogenous manure whereas its true function lies in the formation of humus. Humus is not a single substance. It is an agglomeration of different materials, organic and inorganic, which, when being brought together and mingled are basically modified and transformed to create the basis of soil fertility. Within this process of transformation lies the only correct solution of the disposal of organic wastes. It is an error in principle to treat dry organic wastes and faecal matter in isolation; they must be combined towards the creation of a product which shall be both essentially innocuous and valuable. This is alone accomplished by the application of the ancient principles of composting.

Early in the present century Sir Albert Howard was appointed Imperial Economic Botanist to the Government of India, and at that time malnutrition and starvation were even more prevalent than they are today throughout the whole of that subcontinent. The agricultural policy of the Imperial Government was to increase food production by replacing some of the more primitive practices of the Asiatic Peasant with the more up-to-date methods of European countries. Howard, who was closely associated with the introduction of these innovations, appreciated that the increased demands which they made upon the soils would deplete their content of humus but was convinced that this could be balanced by composting the limited amounts of cattle dung available, augmented by the night soil and other organic wastes accumulating around the villages of India, to produce humus. «Cleaner and healthier villages», wrote Howard in the first book he published on the subject, «will then go hand in hand with heavier crops».

Humus is a complex substance and the factors which contribute to its production are no less complicated. The chemical differences between fresh organic matter and humus are not easily defined and cannot adequately express the essential differences between them. But the relative proportions of carbon and nitrogen provide a convenient and probably adequate measure of the suitability of organic matter for use as a manure. The ratio of carbon to nitrogen in fresh organic matter may be as wide as 500 to 1; in farmyard manure it is around 15 to 1; but in the case of humus it is stable around 10 to 1. The purpose of composting may therefore be described simply as providing for the narrowing of a wide carbon/ /nitrogen ratio to something approaching 10 to 1 before the wastes are added to the soil. The final stages of decomposition taka place in the soil, and a compost may be considered to be mature if it has a carbon/nitrogen ratio of around 20 to 1.

The decomposition of organic wastes is effected by microorganisms which obtain food for growth from the nitrogen complexes in the wastes and energy from the carbonaceous matter, converting the carbohydrates to carbon dioxide and water. When the mass of wastes is well ventilated decomposition is effected by aerobic bacteria but if it is allowed to be compressed during decomposition the interstices between the particles of waste become saturated with carbon dioxide and aerobic bacteria give way to anaerobic. The anaerobic organisms act slowly obtaining energy from sulphur and nitrogen compounds to produce foulsmelling gases such as sulphuretted hydrogen. It is therefore essential for the rapid and inoffensive decomposition of the wastes that air is kept circulating constantly throughout the mass.

It has been established beyond doubt that the microbiological environment existing within a mass of organic wastes decomposing under aerobic conditions is so unfavourable to pathogenic organisms that none can survive. In addition to this natural safeguard, the high temperatures developed with aerobic composting offer further protection. No pathogenic organism is likely to survive when subjected to temperatures of around 160°F. for a few minutes or 120°F. for several days and temperatures within that range and over that period of time are characteristic of all established aerobic composting methods.

The process of composting which Howard devised has taken the name «Indore» from the Indian State where he did most of his work and the principles which he enunciated were so fundamental and so scientifically true that no difficulty has been found in applying the process which he worked out to the treatment of domestic wastes. In practice the process has also proved to be flexible and is equally effective when applied to the treatment of small or large quantities of waste organic matter of widely ranging composition.

In 1929 the Dutch Government, anxious to obtain supplies of humus to enrich land reclaimed from the sea and to maintain the exceptional rate of production in existing land, formed the Refuse Disposal Company Limited (V.A.M.) which was charged with the duty of converting town wastes the composts. In 1931, the company set up their plant at Wyster, which now treats 163,000 tons of refuse a year brought in by rail from the Hague and other cities. At the plant the crude refuse is tipped from the wagons into composting cells which are about six metres in depth, constructed along both sides of the rails. As the refuse is tipped each layer is sprayed with water. The moistened refuse is then allowed to ferment in the cells for a period of four to eight months when it is dug out by travelling grab crane and transferred to a building where the finer materials are screened out and the larger and more durable organic materials are pulverised in hammer-mills. Since 1950 nine new composting plants have been constructed in Holland. All of these are more thoroughly mechanised than the original plant at Wyster and provide for extracting large inorganic items from the refuse before subjecting the remaining organic materials to the composting process. This residual organic matter inevitably varies widely in character. The limits of variation are the durable lignins of all types of wood and the green vegetable wastes from the kitchen. Within these extremes there is a wide variety of matter all of which decompose at varying rates and outside this range of cellular matter there are phosphatic wastes (bones and horn) and nitrogenous wastes (offal). To ensure a uniform decomposition of such a variety of matter, they must be intimately mixed and a large surface area of the more durable materials exposed to the active bacteria. This is accomplished by pulverisation which has become, in one form or another, an integral part of municipal composting.

An object in treating domestic wastes by composting is to speed up the rate of decomposition and some attempts have been made to achieve this by adding cultures containing special bacteria, for which it is claimed that they will reduce the composting time to a few days or even hours. The use of laboratory-cultured organisms in this way, is contrary to any scientific conception of the composting process. The active bacteria are indigenous to the wastes themselves and need not be added. Each group can survive the wide range of environmental changes which constantly take place within a decomposing mass. One organism can produce a million others within five hours, but no group is capable of multiplying at a faster rate than its developing environment will allow. The introduction of organisms into an unfavourable environment must therefore be abortive and interference with this or other biological factors cannot be justified.

No more can be done than create an environment within the mass that is favourable to the most active bacteria.

When the wastes to be treated have been moistened to contain between 40 and 60 per cent of water which can be achieved by the addition of faecal matter in any form, thereby improving the content of nitrogen in the wastes and effecting hygienic disposal, optimum conditions for decomposition are obtained merely by ensuring the free circulation of air within the mass. During the process of decomposition the mass of the wastes contracts which tends to exclude air, and the wastes have to be agitated or turned over in order to restore optimum conditions. The more frequently this is done the more quickly is a mature compost produced. When small quantities of waste matter are being treated this operation can be performed manually or by means of simple mechanical appliances. With bigger masses however the problem becomes more complex and it is here that the greatest difficulty in applying the process of composting to the treatment of organic wastes on a large scale lies.

The first move towards the thorough mechanisation of composting restricted the volume of wastes being treated at one time. This reduced the distance which air had to penetrate into the centre of the mass and allowed simple turning mechanisms to be installed. The elevation of the wastes to be automatically dropped through a series of platforms built into a tower is a later development and is now a characteristic feature of several mechanised composting methods. The Dano System, developed by a Danish Engineering firm, is unique in that it combines all the essential provisions in composting in one operation. The crude organic wastes are fed into a slowly rotating cylinder with liquid faecal matter or water added. Air is injected into the wastes under low pressure through a series of ducts which run through the whole length of the cylinder. With the addition of moisture and air, aerobic fermentation is stimulated while within the rotating cylinder the decomposing surfaces of the organic matter are rubbed off, thereby exposing new surfaces to the action of the bacteria. Thus the moistened wastes effect their own mixing and grinding within the cylinder. The composted materials are finally separated from inert matter by screening. With suitable materials such a plant can produce mature compost within a period of five days, but with the normal run of dry organic wastes contained in domestic refuse which have a high carbonaceous content, an additional maturing period of two to three weeks is required to produce a compost which can be used immediately for feeding crops.

Since the end of the war, composting plants of the types shortly described above have sprung up in almost every country in the world. Nearly 50 Dano Plants alone have been erected in seventeen different countries. As would be expected some difficulties and disappointments have been experienced in the development of these new processes. But it can now be said with confidence that simple composting processes and highly mechanised systems have now moved beyond the experimental stage so that all public health authorities may now make use of composting to meet their problems in organic waste disposal and at the same time allow them to contribute to the production of food by increasing the fertility of the soils around them. The cost of treating organic wastes by these new composting methods is no more than the older and less efficient methods of disposal by destruction. Waters polluted with sewage; sludge lagooning with all its inefficiency and inconvenience; air pollution from expensive refuse destructors; none of these evils need be tolerated further if public health authorities have the will and determination to conserve organic wastes for their rightful purpose of maintaining soil fertility.

PRESENT-DAY PROBLEMS IN COMPOSTING

O. JAAG [1]

The problem of the composting of solid household refuse and other solid organic waste material from towns and villages as well as from trade, industrial and municipal enterprises must be considered from two different points of view. First, the task of public cleansing requires the disposal of the waste material in an aesthetic, hygienic and economic way, avoiding any danger for surface and underground waters and for the integrity of the landscape. Second, the values comprised in the immense quantities of garbage and refuse produced day by day, should be recovered as completely as possible and made available for the country's economy.

These values are represented by the heat capacity of garbage and refuse and by those organic components which can be utilized as conditioners of soils poor in humus and in sandy and acid, marshy areas. In addition nutrient elements such as nitrogen and phosphorus, trace elements and growth stimulante are introduced into the soil. Application of composted garbage in an adequate way can thus improve agricultural production.

With regard to the effective disposal of city wastes collected, two quite opposite methods are being applied in Europe at the present time. On the one hand, waste incineration has proved effective in larger urban communities, particularly when waste is disposed of hygienically in incinerators yielding heat and power, which makes it an economic proposition. On the other hand, waste can be composted. In America gas-producing rotting installations have been developed, but as far as we know, such equipment does not exist in Europe.

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[1] The author did not attend the meeting.

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With the incineration of garbage, tremendous amounts of organic material are lost which could be used advantageously in plant cultivation, particularly in flower and vegetable growing, in vineyards, in forestry, in land reclamation schemes; moreover, such organic material is also urgently needed in some countries or municipalities.

In nearly all civilized countries in the world, for hundreds, even for thousands of years, compost has been produced from solid waste of human settlements by simple methods, thus preserving the fertility of the soil, more particularly in gardening activities. In more recent times, the obtaining of soil-improving organic material became urgent, particularly in the Netherlands, where large areas of barren dunes poor in humus or acid saline soils had to be turned into fertile arable land to increase the farm acreage. In Denmark, France and in other countries, manure available today in smaller and smaller quantities as a consequence of less cattle breeding has to be replaced by other organic substances from various sources. Why not, under the circumstances, make use of the daily city wastes?

Under the pressure of events, particularly in the Netherlands, Denmark, Italy and in the United States, pioneer work kas carried out in the development of rational methods of waste processing. Later on, other countries could benefit by such activities when garbage processing was adapted to local conditions, improved and developed further by additional methods.

More recently, the need for greater amounts of organic substances as available in garbage compost was demonstrated in Germany too, whereas in Switzerland, health and economic reason in particular call for an urgent disposal of garbage, and the compost thus obtained has to find as far-reaching applications as possible.

GARBAGE INCINERATION VERSUS COMPOST

In Europe, garbage incineration and composting are the two essential methods of waste disposal. More recently, whenever in my country a municipality had to discover a final solution to the garbage problem, a bitter struggle arose between the respective supporters of incineration and composting. In view of the opposite aims of the population concerned on the one hand and industry on the other hand, this struggle is understandable; yet, it is not always conducted in the most effective and intelligent manner, and therefore does not always lead to the best possible solution.

However, an adequate objective result should be attainable in the very interests of the municipality concerned, provided the local data are considered per se, outside any general beliefs or private interests, and discussed with full account being taken of any specific local condition.

Any effort to induce a municipality to build an incinerator, for example, is to be considered irrelevant, when the number of inhabitants to be covered is not large enough to make such a proposal economical and it is just as foolish to set up a composting plant when the sale of the final product of the compost is not fully assured.

Such planning can only bring about results generally satisfactory if all group concerned compare data in the health, economic technical and organizational fields and study them objectively. Such a procedure is sure to safeguard any municipality against later disappointments or reproachful considerations.

So far, large and medium sized townships primarily have decided in favour of garbage incineration and have had favourable experiences. However, under special circumstances, when the need for humus-forming substances is great, major cities like the Hague, Delft, Groningen in the Netherlands, Heidelberg, Banden-Baden and Duisburg in Germany have gone over to garbage composting. Over 200,000 tons of finished compost are produced annually in 12 Dutch composting plants (processing 25 % of the total garbage); 100,000 tons are produced in the Wijster plant alone, processing the garbage of the Hague, Groningen and Zandvoort (800,000 inhabitants).

The waste disposal or garbage processing system to be chosen by a municipality must be economically feasible, which does not mean that it must be self-supporting or even become a source of income. As a rule, this is impossible with any system whatsoever. Every kind of waste disposal will remain a financial burden for the municipality which builds and runs it. However, good, sound planning should provide a low cost solution while taking into account all justified interests.

The cost of a garbage incineration plant falls as the number of inhabitants covered is served. In Switzerland a plant serving

500,000 inhabitants would cost Sw. Fr. 20 — per inhabitant, whereas the cost would be Sw. Fr. 50 — if 75,000 inhabitants only are served.

The cost of refuse composting plants generally are lower, even when a relatively small number of inhabitants are to be served. We have calculated that such a plant can be built up at about Sw. Fr. 10 — per inhabitant if 100,000 or more people have to be served. The cost would rise up to about Sw. Fr. 45 — if 4,000 to 5,000 inhabitants only are to be served.

It is more difficult to compare the operation costs for various plants because the calculations are made once with and once without capital service and because the credit and capital conditions as well as the rates of interest vary very much in different countries. Generally, however, the operation costs for incinerators are several times higher than those for composting plants. At present we are working on such a comparison of the operation costs. It would be too early, however, to make any definite statements at his juncture.

In the opinion of an important enterprise for garbage incinerators, at the present time and for economic reasons, such a plant can not be recommended to a municipality, unless it is to serve more than 50,000 inhabitants. However, by grouping adequately a number of smaller municipalities, it might be possible to serve enough inhabitants justify the erection and the economic operation of an incinerator.

Garbage incinerators, such as those in operation in Europe, for instance in the Belgian cities of Brussels and Monceau, in Hamburg (2 plants), Stockholm, Paris (3 plants), Toulouse, Nancy, Roubaix, Bordeaux, in Rotterdam, Amsterdam, Dortrecht, Glasgow, Sheffield, in the Danish towns of Gjentofte and Frederiksberg, and also in the cities of Zurich, Basle, Berne, Lausanne, in the spa of Davos in Switzerland, are the choice solution when no economic use of garbage could be found. In Germany, their number has fallen recently to two incinerators (Hamburg). Also in overseas countries, for example in Calcutta, garbage incinerators were shut down for economic reasons, whereas very recently, the cities of Lausanne and Bernin Switzerland have introduced garbage incineration because the chances of garbage compost disposal in our country were estimated to be very slight.

We shall now discuss garbage processing and composting only, taking several current problems and the results of recent investigations into consideration.

ORIGIN, COMPOSTING, QUANTITIES AND VALUE OF SOLID WASTE

Solid waste is produced:

- *a*) in households and on farms household waste;
- *b*) in cleaning streets and public places, market halls and places; street refuse;
- *c*) in slaughter houses and other municipal enterprises;
- *d*) in many processing enterprises in trade and industry. In most cases the removal of such waste does not fall upon the municipality but on the individual enterprise;
- *e*) in hospitals, chemical, biological and technical laboratories where radioactive isotopes are used or in nuclear reactors and, in the near future, also in atomic power plants: atomic waste, requiring special treatment. Such waste material is to be deposited neither in municipal garbage processing plants, nor in open spaces.

On the basis of European and American conditions, Pöpel has calculated a mean value of 1.10 kg garbage per inhabitant and per day. This quantity differs greatly from country to country and from day to day. However, the minimum average for the cities and countries concerned is only 0.23, the maximum, however, is 2.55 kg per inhabitant and per day. Logically, countries in the temperate and cold zones where houses have to be heated with coal primarily produce larger quantities of waste than countries in warm areas and countries using fuel-oil, gas or electricity for heating purposes (as for instance Switzerland).

It is beyond the scope of thin paper to explain here the various systems of garbage processing. In the paper, distributed among the conference members you will find a brief description of some procedures provided with illustrations and operation diagrams, let me only mention them here briefly.

Beccari (Italy) has found a simple composting method, recently improved on in various ways by Verdier and Peter, for instance. It consists in simply storing the material in well-ventilated containers over grates. The fermentation cells require neither complicated machinery nor constant maintenance. Removal of coarse solid material and sieving of the fine garbage are done manually. After rotting for 40 to 50 days, the material is separated from coarse solid matter and the fine residual material is sieved. Such equipment is still in operation to-day, mainly in France (Avignon and Cannes) and in Italy.

In the older Egsetor method, of the Danish firm DANO, the unsorted crude garbage is carried from the collecting container on a conveyor belt to a rotating sieve-drum where it is crushed while artificially wetted, and the fine garbage is separated from the coarse material. The fine material is then heaped up. Bits of iron are sorted out by magnets and the other metals are separated; clogging material, for example, wood, rubber, textiles, non-ferrous metals, bones, etc., are carried to the waste dumps. The crude compost contains considerable quantities of glass splinters, which naturally lower its value and also reduce its possible use in gardening and agriculture. Such DANO-Egsetor plants are found in operation in Denmark, Sweden, Germany and Switzerland.

Pre-crushing of organic materials in household waste can also occur in a vertical hammer-mill, as, for example, in the Dorr Sieve Rasp, where the decayable crude waste is crushed and freed from the clogging material. In order to make the glass splinters harmless, a crush-roller is connected to the rasp. This system has been used particularly in the Netherlands and also in Germany (Heidelberg).

A new system designed by the Swiss machine firm Gebr. Bühler, Uzwil, processes the unsorted crude garbage by means of a hammer-mill and a crush-roller, yielding in this way a splinterless product. Bulby clogging material, even baled garden refuse, can be processed in this very robust and highly efficient plant.

In the Baden-Baden system refined garbage is mixed with given quantities of thick sludge evaporated in the sludge beds of the central sewage treatment plant. The quantity of sludge to be added depends on its water content.

The addition of digested sludge enhances the quality of the compost. Actually, the garbage, which is relatively poor in vegetable nutrients, is improved in respect of nitrogen and phosphoric acid compounds and trace elements; moreover, the C/N ratio is improved too.

Immediately after the stacking of the garbage, there is a significant rise of temperature inside the heaps, so that the temperature of the garbage may rise to 75°C. Then, however, the temperature curve falls slowly, which allows us to assume that a first stage in garbage maturing has come to an end after relatively few days. This induced the DANO firm to alter the first phase of the decomposition and mechanise it, while maintaining artificially the major factors of dampness and ventilation. Furthermore, the temperature can be checked through a regulated air inflow. Thus the new DANO-Biostabilizer process was developed. The heat produced by the biochemical decomposition in the stacks kills the human and animal pathogenic germs, the worm eggs, and also the weed seeds contained in the sludge or garbage, so that the final product — the garbage-sludge compost — is perfect in every respect.

Recently, the biostabilizer method has been used for the joint treatment of garbage and fresh (Edinburgh) or rotted sludge (Rüschlikon, Chur, Duisburg, among others) whereby the value of the product is greatly increased, so that such garbage-sludge compost is now more or less equal to stable manure with regard to its soil improving and fertilising effect.

A town or a group of comparatively small municipalities find it advantageous, of course, to complete the entire processing operation in one step.

Our investigations have shown that the crude garbage we use with its average water content of about 30 % can be processed together with sludge containing about 70 to 75 % of water in such a manner that the total amounts of the garbage collected in the respective areas of the municipality can always be processed with the total quantity of sludge collected.

There are two possible basic uses for crushed refuse:

a) For heat packings, in gardening, whereby, as when adding horse manure, the heat developed in the rotting process is used to warm up the soil in plant growing.

b) Distribution of fully rotted garbage compost in gardening and vegetable growing, in vineyeards and in forestry as well as in agriculture, to maintain or enrich the organic substances or to improve the soil structure, and

> also partly to provide the soil with the required essential nutrients for the cultivated plant as well as to introduce the trace elements necessary to further growth.

The appropriate treatment of garbage and of the sludge/garbage mixture as a mean to improve and fertilise the soil has been discussed in the distributed paper.

The garbage compost is certainly a desirable soil improving agent but not a fertiliser in the real sense of the word. Therefore opinion about the value of compost vary greatly. Whereas some people consider it to be responsible for almost miraculous effects, other circles assumed to be open to expert understanding of these questions meet it with utter lack of interest or accept it but conditionally as a soil improving agent.

Gardeners and vegetable growers are in need of large quantities of garbage compost, as appears from the rapid sales of the product at all existing processing plants. At times, the compost has to be rationed, like food during the war, and frequently the stacks are depleted long before the decomposition process has come to an end. If the compost still proves to be somewhat deficient, containing, say, certain amounts of glass splinters, or showing too alkaline reaction, or an inadequate degree of maturation, the competent authorities should not dismiss the utilisation of garbage compost as result of these shortcomings, but rather assist in overcoming the difficulties and give the garbage compost those qualities demanded in practice. Problems exist only to be solved!

In our paper we have mentioned some examples of the various possible utilisations of garbage. Success has been achieved especially in forestry in the Netherlands and in Germany. Chief forester Cosack, Düsseldorf, writes about it: «This is actually a fantastic success on a sandy soil previously utterly poor and barren».

In contraste to these results, there was practically no improvement in the surfaces treated with mineral fertilisers only, and growth started only after garbage compost was spread over these areas. This demonstrates that mineral fertilisers can be successfully used only on soils provided with adequate amounts of humus material.

Yet, there are not in every country large areas of duned, sand, or acid heath soils, as in the Netherlands or in Germany; in spite of this, those countries must find a solution to the problem of effective municipal garbage disposal.

Even in these countries, large amounts of garbage and garbage/sludge compost are used in forests. Tests caried out so far in the municipal forests of Zurich have demonstrated that the addition of a mixture of municipal garbage and sewage sludge shortens the necessary rotting period to a large extent. Furthermore, this addition of garbage and sludge improves structurally and qualitatively the finished compost since it entails a rise in its plant nutrient content. It has been calculated that the total amount of household refuse in Switzerland would scarcely suffice to produce the amounts of compost required in forest nurseries in the next ten years.

We are already faced with the problem of using garbage and garbage/sludge compost for the re-afforestation of forest areas burnt--out, or destroyed by avalanches, and to sped up the reconversion of de-forested pasture land in the protective mountain forest.

According to the latest results of the experiments conducted by our specialists, young plants grown in compost soil can be transplanted 1 to 2 years earlier than those from untreated nurseries.

Furthermore we feel that garbage compost may find very significant possibilities of application in vineyards in the near future, since Mr. Klenk, Germany, could prove that, on steep slopes, much less damage was caused to the soil by downpours washing out the land, than where stable manure had been used: the addition of compost prevents the formation of crusts on soils poor in humus, improves the water retention capacity and thereby the biological activity of the soil and simultaneously the micro-climate of the vines themselves.

As I mentioned in my paper, garbage compost has recently found a quite unexpected new field of utilisation in chicken breeding. The usual sand and peet litter in chicken houses, which must be renewed every week or every other week may advantageously be replaced by a mixture of compost and other litter material. After successful experiments with this new method, so rapidly accepted in progressive chicken breeding in the Netherlands, Mr. Teensma could recently report that it had already become common practice through out the country.

The floor of the chicken house is covered with a 30-50 cm thick layer of garbage compost, straw, corn cobs, wood shavings, horse manure and peet litter. On this foundation, the dampness and ventilation of which are kept under constant control, and which is renewed only once in every 3 or 4 years, the chicken live out their entire life. Through the decomposition processes in the litter material, heat is continuously produced, which is an advantage in winter, as hens and chicken no longer need to flock together to keep warm. This protects the animals from infectious diseases and suffocation. Cannibalism among the hens, which used to cause great damage, also declined to a minimum since the floor is covered with garbage litter.

A new and interesting field has therefore opened up for the utilization of garbage, which helps us to solve the problem of appropriate and profitable utilization of municipal garbage.

INTERNATIONAL COOPERATION

It is a fact today that by means of various methods town garbage can be processed into a valuable soil-improving agent and, by the addition of sewage sludge, into a fertilizer as well. We have tried to explain above that the product obtained through such processing can be used in various ways and play a major role in the economy of our countries, particularly in gardening and vegetable growing, in vineyards and forestry, and, in part, in agriculture too.

Yet we have not failed to point out the many problems which are still to be solved. These problems are more or less the same in all countries. Some basic questions of definition, chemical and structural composition of the garbage compost, the method of analysis which would make it possible to compare the results obtained by research in the individual countries are still to be clarified. Furthermore, the characteristics of good garbage compost must be defined in consideration of its various applications. Experimental applications to plant growing must be carried out to allow us to determine the effects of the compost treatment, the most favourable procedure, the proper timing and the optimum quantities of compost to be used. And finally, the results should be tabulated quantitatively if possible, taking into account amounts, quality and other criteria of crop yields, and set up comparatively for plots treated with compost and plots left untreated. A relationship is to be established between crop yields obtained and plant building and operation costs, costs of transportation and application in the field, etc.

There are also technical problems still to be solved, for instance, total elimination of slag and ashes as well as glass and non-decomposable elements, and finally the adequate turning over of the stacks. It is up to science to study the former problems, it is up to the industry concerned to deal with the latter. Progress can be achieved only if there is close cooperation between science, applied agriculture, industry and municipal administrations. There are a great many unsolved problems indeed!

When we came to take up these problems in Switzerland we did not want to reconsider those problems which had already been solved in other countries. We also wanted to avoid taking the same route in our search for knowledge as others had taken in order to reach their goal.

Therefore, in cooperation with the German working group for water and sewage research and with Mr. Weststrate, director of the V.A.M. Amsterdam, e invited, in the summer of 1955, the most experienced experts in our field, from 10 countries, o take part in a compresensive exchange of ideas. The most important result of this conference held at the Federal Institute of Technology (Eidgenössische technische Hochschule) in Zurich can certainly be seen in the unanimously adopted resolution to create an international working group on garbage research, entrusted with the particular task of coordinating the research programmes in the individual countries in view of cooperation at the European level. Furthermore, this working group is to formulate new problems and to make the results of research available as rapidly as possible to the groups concerned in all participating countries.

Since that date working groups have been formed in Germany, in the Netherlands, in France and in Switzerland, they tackle particular problems and also contribute jointly to the expenses of the Secretariat.

Our Secretariat has already published three information bulletins; the last one was issued for our present conference. It is published in German and in French and we have good hopes of being able to bring out in the near future an English edition. Daily inquiries from all over the world for subscription to the bulletin and requests for past issues show the great interest taken in this information bulletin, and the first two papers were soon out of print so that a new edition became necessary.

We hope in this way to be able to make a contribution in this important field which is vitally related to the general tasks of urban cleansing.

PROBLEMS OF WATER SUPPLY IN THE TROPICS

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The problems of water supply in the tropics are fundamentally no different than those of other portions of the earth. Many of these problems have resulted from a slowness to realize that water is not only a day-to-day necessity but also a national resource only less important than the men and women who are necessary to the development and prosperity of a nation.

The average citizen takes water so much for granted that he rarely considers its importance to himself, his family, his community and his country. It is, he knows, a necessity for human existence but he rarely thinks that large cities could not exist without adequate supplies for such purposes as domestic and commercial uses and fire fighting and other public purposes. Industries of many types must have it in large quantities. It carries away liquid wastes. Agriculture must have it. It is used for water power, sometimes over and over again. Navigation needs it. In some fortunate areas it is important for recreation. In others it provides an important source of food. It is all derived from the rainfall, which in some areas is relatively constant in amount but in others is widely variable from month to month and year to year.

Blindness to the need for study of water resources and the problems involved in making the available water useful has not been confined to tropical countries. It has usually been neglected everywhere until stern necessity has wakened governmental authorities to the need for immediate action. Then the results of neglect become

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[1] The author did not attend the meeting.

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painfully apparent for it then becomes necessary to design and construct works for the collection, storage and use of this precious commodity, water, with insufficient basic data. The engineer knows that this may mean danger of expensive failures and at least uncertainties in design and waste of money.

Engineers, who are accustomed to thinking in terms of the future, know what should be done, and if their single voices are of no avail, then through local organizations they should bring pressure to bear upon the national authorities who control policies and finances. The ideal solution would be for any nation to have, so far as humanly possible, for all of its area, the data concerning rainfall, and its distribution annually and monthly; stream flows, minimum, maximum and average, annually, monthly and daily; and also the amount and location of underground water.

Some of the more industrially advanced countries have gone far in obtaining of such data. Many have made little or no progress and some of these are tropical countries. The reason for this is that in those countries industries have been slow in developing, populations have been sparse in many areas and the necessary engineering personnel has been lacking. The situation, however, is changing. Better communications and transportation are awakening the peoples of all classes and areas and the resulting progress will soon place enormous demands upon all national resources, including water.

The obvious answer is immediate steps toward obtaining basic information, compiling it, analyzing it and making it available. It should be pointed out that basic statistics are of no value if they are buried in the dusty files of some office until their very whereabouts is forgotten. It should always be recognized that adequate information as to ample water supplies is a strong attractive force to industries that are looking for a place to locate. The result of this can be quicker development of areas undeveloped now.

I should point out the necessity for considering legal questions arising in connection with water. This is not an engineering problem but it can enter into or prevent proper solutions of those problems. Water laws established a hundred years ago may not be applicable to present day conditions. Accordingly, at the beginning of a comprehensive water conservation or study program the existing water laws should be examined and if necessary modified to allow proper development of a conservation and use program.

Basic hydrological data are needed and they cannot be obtained in a year nor in ten years. The fluctuations in rainfall and other climatic conditions require years of observations to eliminate large errors, although a ten-year record is, of course, helpful. A beginning is collection of rainfall data, the primary source of all water supplies Rainfall is predictable only to the extent that its probable variations in any one area are known. Therefore, many rainfall data must be collected and analyzed. In urban areas short-time intensities, as well as total rainfall, are needed for storm drain design.

Rainfall data, while important, are not sufficient. We must know what proportion reaches the streams and underground formations. This calls for somewhat more elaborate and expensive procedures than obtaining rainfall data. Stream gaging stations will be needed at strategic points with regular readings of river stages. This may present real difficulty in a sparsely populated area where communications are difficult. Again concurrent analyses of the data obtained are needed, not only for the development of basic information, but to uncover gaps in knowledge that can be filled before areal development becomes an actual fact to be faced and its water problems must be solved.

Funds, of course, are not always available in the abundance which may be desirable. Hence it may be necessary to proceed in piecemeal fashion. In other words, it may be necessary to begin the information-obtaining program in certain circumscribed areas. If this is the case the river basin should be the unit of study and those drainage basins which are of special importance would naturally be chosen first.

Underground waters can be of great importance in the development of an area. For municipal and village supplies they are preferable to surface waters. Exploring for their occurrence will require many test borings and skilled observations of the underground geological formations and necessitate the combined services of field and office engineers and geologists.

While the quantity of water available in any area is of paramount importance, quality is also an important consideration. In health work we think first of quality with respect to disease. In an undeveloped region where sewers are nonexistent and population is sparse, sanitary quality may not be a problem. As the water is used and reused, however, for such purposes as industries and municipal water supplies, wastes of various kinds will enter the streams and the waters of the river basin will become progressively polluted as they proceed toward its mouth, except in so far as natural purification remedies conditions. Where pollution has already developed studies should be inaugurated that will indicate the self-purification capacity of the river in question as an important step toward its maximum beneficial use as a source of supply and also as a carrier of wastes that must be disposed of.

The sanitary quality of water is important but we must also consider its chemical and physical qualities. Some impurities, as turbidity, can be rather easily removed by modern methods of water treatment and the same can be said for disease-producing organisms. Some chemical impurities, however, may make a water practically unusable. This applies more especially to ground waters and accordingly any investigation of ground waters must be accompanied by chemical analyses to determine their dissolved impurities and thus to obtain an understanding of the effects of those impurities on proposed or possible uses of the waters. A high saline content, for example, makes water unusable for irrigation. Too great hardness may make it unusable for domestic purposes without treatment and, even if treatment is possible, its cost may be very high.

Surface water is generally usable for most industrial purposes and also for municipal or domestic use, after treatment to make it safe. But there are exceptions, particularly during dry weather, when flow derived from underground sources may increase the mineral content to undesirably high amounts. Therefore, the appraisal of value of water, whether surface or underground, requires the services of the chemist, as well as the engineer and geologist.

So far I have been advocating the acquisition of data in that branch of engineering science which the civil engineer calls hydrology. I have been emphasizing its importance to the development of a country or a definite area of that country. As a sanitary engineer I will take the opportunity to speak of a problem which is also neglected to some extent everywhere but even more in the tropical regions that I have had the privilege of observing.

Civil and sanitary engineers have shown professional awareness of the problems of water supply and sewerage of the large cities but have, like too many official health agencies, tended to neglect the problems of rural sanitation. Most of the large cities of countries located in tropical climates have established water supplies which reflect the technical excellence of the engineers of those countries and the progressiveness of the national and local governmental authorities. I would say that the present problem of those cities is to keep up with the demand for water which characterizes the present-day rapid growth of cities.

The field to which my remarks will mostly apply is that of rural sanitation. This includes individual homes, and the villages and small towns which have not shared in the progress toward better water supplies to the extent that the large cities have.

We should recognize that water supplies, whether large or small, urban or rural, should be under engineering direction in all the necessary phases of investigation, planning, construction, and maintenance. This is not easy to obtain. In the first place, engineers trained and experienced in sanitary engineering are too few in numbers, considering the great needs of their services, and, in the second place, their services are expensive. Furthermore, from their professional viewpoint, engineering work on large projects is more attractive and leads to professional recognition and better monetary rewards. It requires dedicated men to fill what appear to be minor positions in bureaus and ministries of health at the prevailing low salaries and with their professional rewards principally confined to the satisfaction of raising the level of health of the rural and small-city dweller.

The importance of developing rural water supplies cannot be overestimated. I need not, to this group, indicate the numerous and important water-borne diseases which safe water supplies will prevent but I will point out that improvement in water supplies is a first step, easily accepted by the population benefited, in the campaign of rural health improvement. From there on community civic progress, and health improvement in other directions, should be easier to establish and develop. The next step, for example, will usually be better and safer methods of excreta disposal. More water for personal cleanliness will be followed by better food sanitation and the general well-being of the individual and the community will be improved.

Water supplies of small villages, which have not had professional guidance, have usually developed as the result of the ideas of completely uninformed local people with the unhappy results that the water may be dangerous and invariably is insufficient in amount for sewerage and the other amenities of modern life. Or, no attempt has been made for development of a community supply with the even more undesirable alternative that people carry water from streams or unprotected wells or springs. Water so obtained will be inadequate in amount for personal cleanliness and it may also, and probably will, be dangerous.

The answer is obvious, sanitary engineering knowledge as applicable to rural conditions must be made available to the rural and village citizens as well as to the dweller in the nation's capital or other large cities. Many countries have been slow to see the importance of the rural citizen in their economic life. At the beginning of this paper, I pointed out that water was second only as a national resource of a country to its manpower and I am sure no one will disagree. There will be complete agreement also that a safe water is also a conserving agent for that manpower through reduction of the death rate and decrease in water-borne but non-fatal disease. A healthy rural and village population will produce more foodstuffs and other wealth for the nation. Engineering has a proud record in the development of material resources for the use of man. In so far as sanitary engineering conserves lives and health it can boast that it contributes directly to the development of manpower and thus indirectly to other material development.

So far I have mentioned only the establishment of rural and village water supplies. That is only part of the problem. Rural and village dwellers, including the village administrative officials, are not accustomed to maintaining or managing an enterprise even as simple as a small water supply. Their acquaintance with mechanical matters will be slight and they are not accustomed to planning for emergencies or any future program. Supplies and replacements will be difficult to obtain. If collection and disbursement of funds are involved it means that the money will not be properly budgeted or expended to maintain the water works. The inevitable result is that in a few years the system is highly inefficient and inadequate or it may even be completely inoperative.

The answer to this problem is, of course, some method of inspection and control. This involves a large organization on a national basis well staffed with qualified engineers and other technical assistance. It will be costly, but it is necessary to an adequate solution of one of the important problems of water supply in tropical countries.

Technological progress on a worldwide scale is characteristic of the present day. To most people, and I fear to many government authorities, this only means more factories, more mines, more oil wells, more and bigger hotels and apartment houses and unprecedented growth of the bigger cities. The question I am raising, and each of you can answer for your own countries, is sufficient money and technological effort being expended in development of the two fundamental and related resources of any country, manpower and water?

THE PROGRESS OF ENVIRONMENTAL SANITATION IN AFRICA SOUTH OF THE SAHARA

KARL HECHTER-SCHULZ [1]

1. INTRODUCTION

1.1. — This paper is an assessment of the progress of Environmental Sanitation in Africa South of the Sahara. It is based on a review of available literature and the personal experience of African environmental problems which I have obtained from many years of living and working in various parts of the subcontinent.

1.2. — Official information available is scanty and when obtainable is not set out in the best manner. Medical reports are not presented in a uniform way; there is a tendency to generalise and to use loose undefined terms; while statistics and other numerical data which can be used are rare and incomplete. For these reasons the information presented here is selective and the information on which I had to rely has therefore been given my own interpretation.

1.3. — «Environmental Sanitation» has been defined as the control of those factors in many physical environment which exercise or may exercise a deleterious effect on his physical, mental or social well-being. Some of the data selected on which to base the assessment of the progress of environmental sanitation in this paper are: 1) Water Supplies; 2) Wastes Disposal; 3) Housing and Town Planning; 4) Personnel and Organisation; and 5) Vector-Borne Disease Control; and because of the important influence that demography has on the environment, data on this as well as on some economic facts are also presented for consideration.

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1.4. — The environmental sanitation problems of Africa South of the Sahara are tied up with 1) the increasing use of irrigation, 2) the increasing birth rate and decreasing death rate 3) the over-expanding industrialisation without concommittant social planning, and 4) the lack of educational services.

1.5. — The role of the Public Health Engineer or Sanitarian has not yet been defined in terms of meeting the challenge of human fertility in Africa, although the exercise of his science has contributed most to aggravate the problem by lowering the death rates.

2. DEMOGRAPHIC FACTORS

2.1. — The standard of environmental sanitation cannot be divorced from the economic and social standards prevailing in any country nor from the demographic trends now in evidence on the African subcontinent be disregarded. As in all underdeveloped countries, one of the major African problems is that resulting from human fertility. The tribal African has not yet grown out of the mental stage where each child born is considered a financial asset; boys as labour units for communal efforts: and girls for marriage dowry. With the rapid industrialisation of the subcontinent this tenet is a complicating factor of the greatest importance. As modes of life change from communal agricultural living in rural areas to overcrowded slum conditions in rapid-growing industrial areas population growth must be considered along with the traditional insanitary practices of the African as the foremost problem. The traditinal form of itinerant labour brings its own special problems as well.

2.2. — Africa, consisting of nearly one quarter of the world's land surface is inhabited by only about 7 % of the world's population. The population consists mainly of about 200,000,000 indiginous people, with about 5 million European and half million Asian settlers. Most of the Europeans are settled in the Union of South Africa, the remainder are dispersed throughout East, Central and West Africa.

2.3. — National income for the Belgian Congo, Northern Rhodesia, and Kenya is probably representative for all tropical

Africa; this is probably not more than $50 per capita; that for the Union of South Africa is about $264. About 50 % of indiginous people are engaged in agriculture. A good number more are engaged in pastoral pursuits, largely self-sufficient tribal and village economy on a subsistance level. In 1949 Africa accounted for about 8 % of the world's total imports and for 5.9 % of its exports. These exports were mainly precious and base metals.

2.3.1. — There has been a decrease in agriculture, forestry and fishing in all countries except Gold Coast (where these activities have increased). In place of these activities there have been considerable increases in mining, building, transport and general industry. Income has grown with this increased activity in all countries; with the exception of Northern Rhodesia where there has been a fall in income level for all occupations. The increase in income levels has been followed by increases in the cost of living. The wage earner constitutes the largest income group in Southern Rhodesia, Belgian Congo and Kenya (40-60 % of total): while income from this group was lowest in the Gold Coast, Nigeria, and Uganda (10-25 % of total). Both public and Governmental expenditure has increased in recent years.

2.4. — The density of African population is closely related to the continent's geography and climate. While the size of the continent is second only to that of the continent of Asia, the human density is only 15.9 per square mile (Asia is 319.3 per square mile) with the greatest density in West Africa (Sierre Leone has 50 per square mile). Only about one fifth of Africa south of the Sahara lies in the temperate zone, the remainder is tropical. In addition there are great arid and semiarid tracts (Kalahari and Namib deserts and the Great and Little Karroo) situated mostly in the more temperate South where human density is insignificant in numbers. The climate is as harshly differentiated. Rainfall varies from tropical abundance of 50 inches or more to a desert precipitation of only a few inches annually. These factors determine not only the population distribution and density in both rural and urban areas, but also what to pursuits can be.

2.4. — While human fertility remains at an extremely high level with families ranging from 6 to 20 in number, mortality rates particularly infantile are being steadily lowered. (Examples are: West Nigeria from 103.9 to 81.8 for the town of Lagos; Sierre Leone from 182 in 1947 to 124.9 in 1955). Population gains due to these factors are extremely difficult to compute, as most African countries have no comprehensive or complete system of vital registration. However, an estimation of population increases for the whole continent has been made by the specialised agencies of the United Nations Organisation. The information given is not considered very accurate, but as this is the best available it is reproduced here, even though it is based on figures for the whole continent of Africa.

Population Estimates and Density in selected years for Africa

Year	Midyear population	Average annual increase %	Persons per mile
1920	136	—	4.6
1930	155	1.3	5.2
1939	175	1.4	5.9
1949	198	1.3	6.7

The greatest increase seems to have taken place in the temperate zone of Southern Africa where the population (mostly indiginous) has nearly doubled itself from 7.8 million in 1920 to 13.5 million in 1949.

2.4.1. — The Public Health Engineer's direct contribution to the control of population growth under present circumstances must be a negative one. Whatever environmental control he is responsible for must steadily worsen the situation, as many of the natural brakes of population level control are amenable to the discipline of sanitary science. The problems arising from decreasing mortality rates with a resultant increase in population pressures in limited localities can therefor only aggravate the present low social standards associated with the indigenous populations of Africa where the authorities have failed to provide general socio-economic uplift at the same time.

3. WATER SUPPLIES

3.1. — An assessment of the progress of providing pure water supplies to all inhabitants of Africa South of the Sahara is most difficult in the absence of detailed information. An estimation of the percentage of population actually obtaining water from pure reticulated systems can only be based on personal experience of the problem together with the scanty information which is available. On this basis it is considered that less than 6 % of the African population (i.e. 12,000,000) enjoy this privilege. This rate varies from about 20 % in the more highly industrialised southern subcontinent to less than 1 % in most areas in the western and northern areas.

3.1.1. — The bulk of the urban and rural population still obtain their domestic supplies from waterholes made on the banks of rivers or streams, and when these dry up, in the stream-beds or from water drains and holes along the road sides. In many areas these water-holes are jealously guarded as family treasures. Water holes are often long distances from the habitations which necesitates water transportation by hand and storage in all manner of containers on the premises. Wells ar not common and may be sunk in geological-unfavourable places.

3.2. — Although the tube-well («Abyssinian Well») has long been known to sanitarians operating in Africa it has not been exploited to its fullest possibilities.

3.2.1. — Progress can be reported in the extension and provision of pure supplies to the larger urban populations with a total of about 450 towns now reticulated in all the subcontinent. As this progress has cost over £ 90,000,000 to supply 227 towns in the Union of Africa alone, it can be conjectured what it would cost to supply water under similar conditions throughout the remainder of the subcontinent.

3.3. — In the Union of South Africa water supplies for the reticulated urban centres are obtained mostly from storage dams built on the rivers, augmented by water obtained from boreholes, springs and catchment areas. In several instances in the semiarid areas of South West Africa supplies are obtained exclusively from tepid mineral springs, and at Walvis Bay a sea-water distillation plant operates to supply the port and ships with pure water. The Rhodesias are extending present supplies to industrial centres to cope with expansion now taking place there. The French have spent 5 million francs on water supplies to various centres in their dependencies in Africa, but as the bulk of population is still rural (despite rapid urbanisation) there still seems to be a tremendous task ahead for them in these areas. In Uganda extensions and improvements to supplies of 12 urban centres and new boreholes (247 boreholes sunk in 1956)

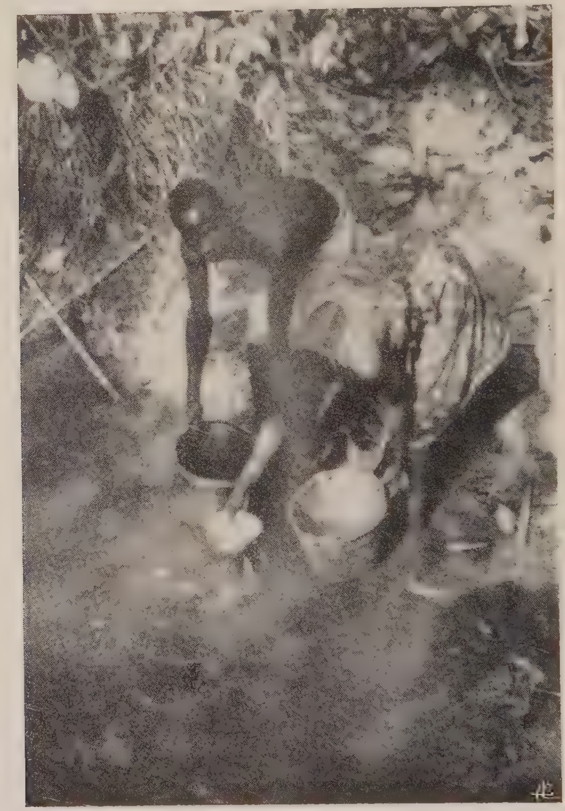

Fig. 1 — A treasured jungle pool of water, shared by a family. Drawer of water must wade into water, with risk of guinea-worm infection

(Photo H. J. Heinz)

is proceeding. There is little progress in Ghana where householders obtain water supplies from dams and waterholes and store this on the premises. In Tanganyika urban water supplies are adequate but may be bacteriologically suspect. The water supply problem in Western Nigeria is reported as becoming increasingly pressing, on account of inadequate staff and material, although urban water systems are being improved and extended and progress made in rural areas by the sinking of numerous tube and other type of wells. In the British High Commission Territories there is a slight improvement in urban supplies but hardly any in rural supplies.

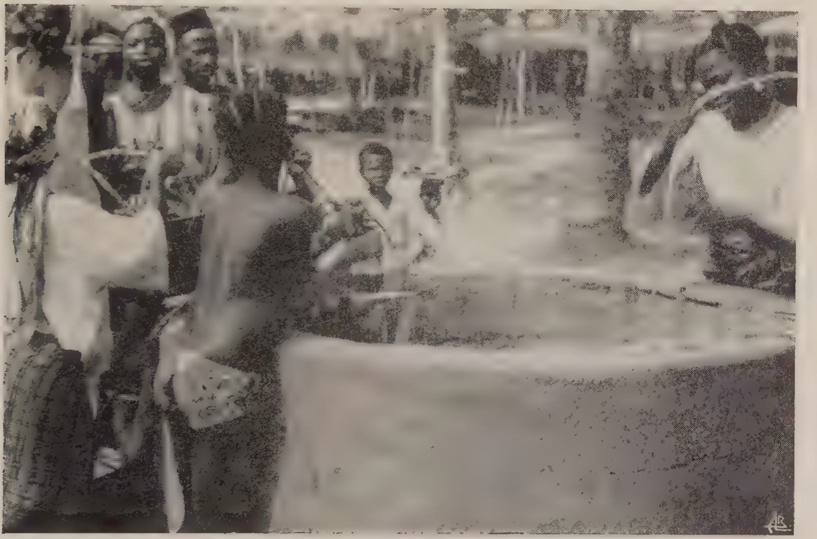

Fig. 2 — A soundly constructed well in a village, simple and effective

(Photo Schulz)

3.4. — No major improvements have been carried out in the remainder of the African states.

3.5. — The intestinal infections which can be spread by contaminated water supplies, amongst which can be considered the typhoid fevers, are common throughout Africa South of the Sahara: while allied infections such as the dysenteries (bacillary and amoebic), are widespread. The spread of gastro-enteritis, which is one of biggest causes of Bantu child mortality in the Union of South Africa, can be partly attributed to transmission through contaminated water supplies.

Ghana reports 10,000 people crippled completely every year from the northern area alone and 90,000 partially so from guinea-worm infection. Other areas of the northern subcontinent also show high rates of infection, with 16 % infection of the Nigerian population. Only in the Northern Uganda area can a claim be made to the reduction of this disease due to improved water supplies.

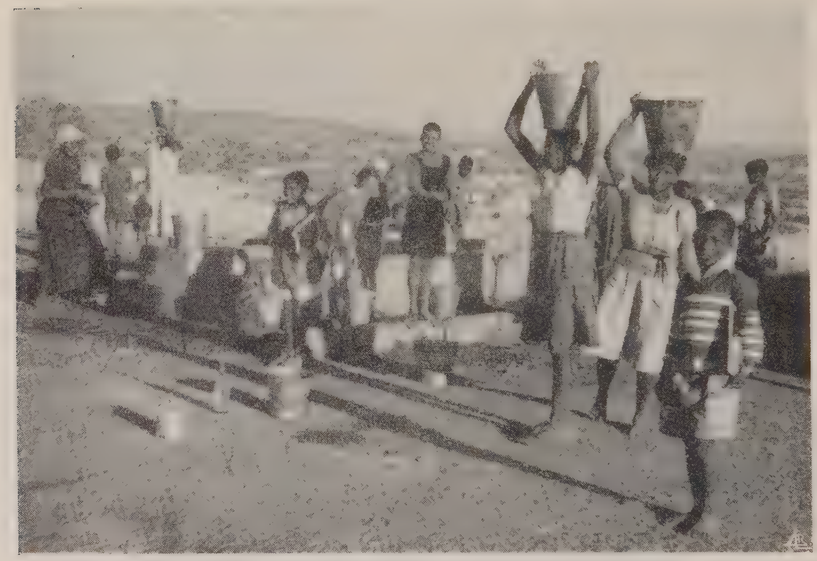

Fig. 3 — Water drawn from good borehole in semidesert some distance away from home, and stored in all types of containers

(Photo Schulz)

Bilharzia has spread so rapidly in the southern areas of the subcontinent that it is rapidly ousting all other tropical and subtropical diseases in importance. This disease is widespread throughout the wet tropical and subtropical regions of the subcontinent, but seems to be fairly well tolerated by the African victim except where heavy physical labour is required of him. A major factor in the spread of bilharzia into new areas has been the elimination of malaria from areas which were formerly dry, but which can now be irrigated from rivers containing the vector snail.

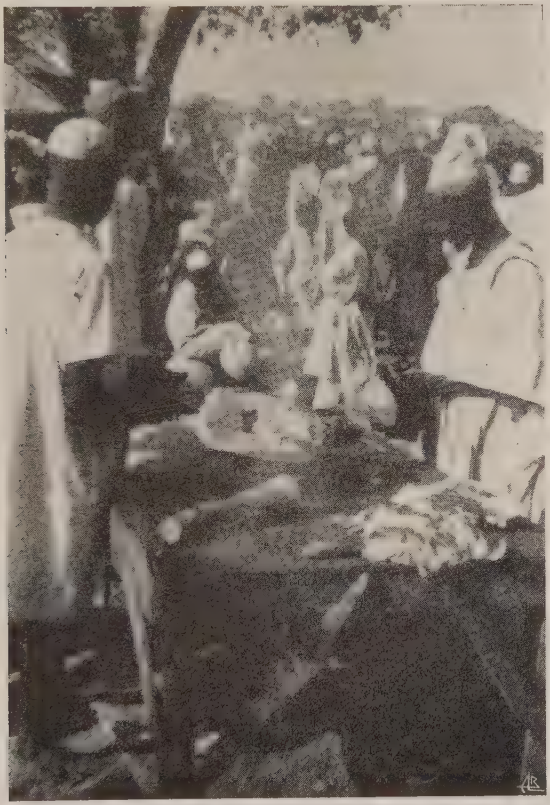

Fig. 4 — Open-air butchershop

(Photo Schulz)

4. WASTES DISPOSAL

4.1. — Wastes disposal, including bodily excretions and trade wastes, consists of the innocuous disposal of all wastes created by man in his business of living. Human faeces disposal is most important everywhere, while domestic refuse and trade wastes disposal is a problem only in the major urban centres where industry is often localised. Animal faeces in the rural areas are generally disposed of by being made into dry cakes and used for fuel.

4.2. — Over much of the subcontinent time has not changed the African habits of disposal of his excreta. Religion and the type of country have an influence on these habits. The native of the savannah still prefers to squat in the open grassland behind a few bushes. Where religion demands that the body remains unseen more success has been obtained when pit latrines with screens have been introduced. In the more tropical areas banana groves serve as a defaecating ground. Under these conditions heavy hookworm infections in the local population are found to be prevalent. In the large rural towns sometimes the only latrines found there serve the few expatriate families, such as the missionaries, traders and handful of government officials. Often regions consisting of many thousands of square miles reserved exclusively for the living of indigenous Africans are found to be without a single latrine. This is true also for extensive farming areas where no attempt is made to supply labourers withe latrine accomodation. Under these conditions the African well becomes heavily contaminated with the continuous human defaecating especially in the vicinity of all towns. Ravines and the heavily wooded sides of water sheds are fouled and in time of rain a good amount of this material is washed into the streams and rivers and other waterplaces used for catching and holding water supplies by the local residents. A further goodly amount of this excreta is eaten by domestic animals kept by the inhabitants — pigs, goats, and chickens being particularly useful in this type of scavanging. Cattle in ranching areas also come into contact with grazing lands fouled in this way, and carry heavy infestations of cystercercosis.

4.3. — In the larger urban centres progress has been made in the provision of waterborne sewage systems depending on the availability of reticulated water supplies and finance. These systems are often coupled with other types of conservancy where conditions are unfavourable for total installations. Sewage disposal varies from modern sludge-digestors with sand-filteration and chlorination of the effluent to broad irrigation of the crude sewage. Only in the southern part of the subcontinent some composting is attempted with the raw sewage, for use as fertilizer. Legislation has lately been introduced here to control the bacteriological standards of such

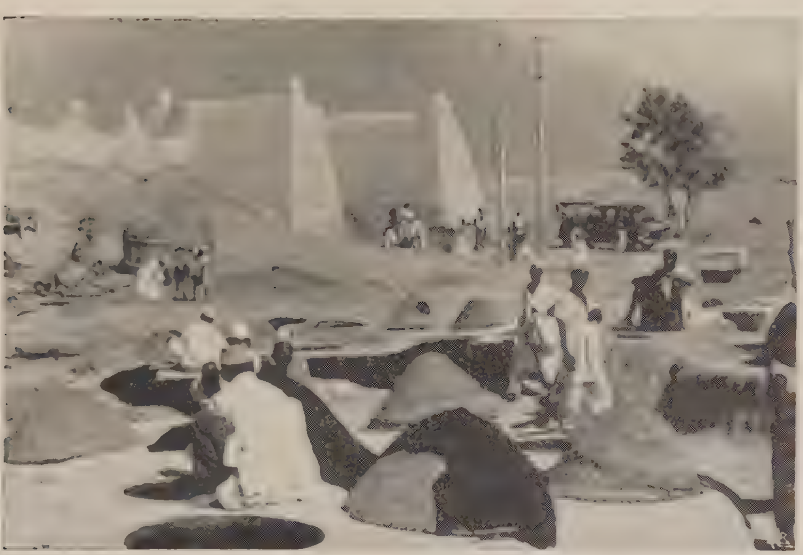

Fig. 5 — Grain storage in subterranean pits prior to sale

(Photo H. J. Heinz)

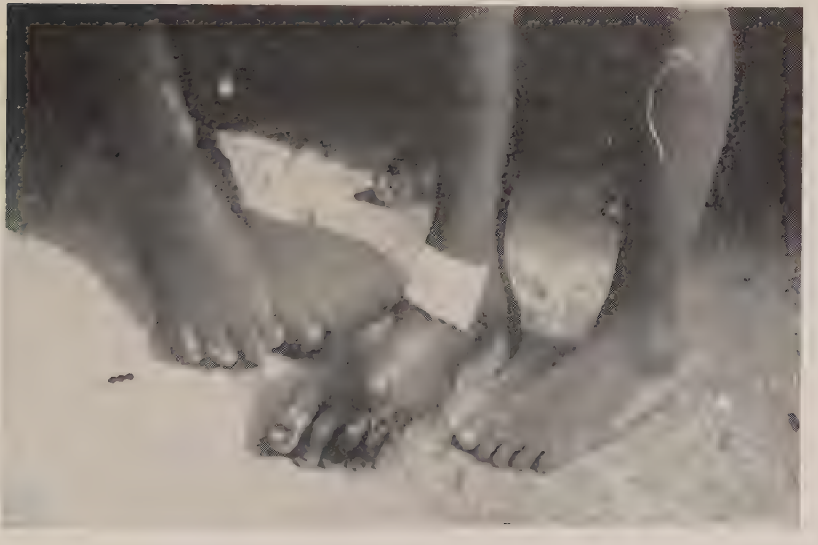

Fig. 6 — Leg lesions of guinea-worm infections

(Photo H. J. Heinz)

compost. Bucket conservancy where this takes place in medium and small urban centres usually is connected with a spillage and disposal nuisance. Only few places use narrow trenching for faeces disposal from buckets: the usual method used is to dump bucket contents in large open pits dug to accomodate several weeks accumulation. In a few places (Zomba the capital of Nyasaland is outstanding example) small septic tanks have been installed and are working successfully, while in other centres they do not function so well, because of ignorant usage and also because no regard has been given to the subsoil water level or the density of the soil in the way of disposal. Outway pits are rarely used.

4.4. — In the Union of South Africa out of 277 towns listed as of some importance, 31 have complete sewerage systems, 46 are part sewered, and 17 have systems under construction. Uganda reports 3 centres completed, while drainage at Kampala will cost £ 554,000 to complete. Ghana is now converting hundreds of miles of earth drains in Accra, which is now surveyed for water-borne sewage: while systems for 3 other centres are also being planned. The standard of sanitation in many towns in Tanganika leaves much to be desired. The bucket system is still the general rule in West Nigeria, where some septic tanks are also in use; the larger towns must still be sewered. In the British High Commission Territories small progress is being made in replacing bucket conservancy with septic tanks in the main urban centres.

4.5. — The high parasitic rate in all Africans and in their food animals grazed on the veld is evidence of the general lack of progress in this aspect of environmental sanitation. Slaughter cattle arriving at abattoirs from certain areas in South Africa are always suspect, and have a high condemnation rate for cystercercosis. Even pigs reared on some well developed farms must sometimes be condemned on slaughter for cystercercosis. Typhoid infection is seasonal in rural areas, while dysentery and diarrhaea play an important part in producing the high infantile mortality. Amoebic dysentery is becoming prevalent in the subtropical areas of Natal and the Transvaal. Uganda reports that taenia and ascaris infestations are widespread; 18 % of all meat slaughtered must first be boiled to destroy the cysts before being sold to the public. Tanganyika had 7,747 hospital admissions for ascaris in 1956. West Nigeria reports 34,473 hospital admissions and attendances for helminthic infections, while other parasitic conditions treated amounted to 27,780. It can be said generally that Africans throughout the subcontinent carry heavy parasitic loads, and that no progress in the control or reduction of these parasitic diseases can be demonstrated in Africa South of the Sahara: except perhaps for the reduction of hookworm infection amongst the labourers of the Witwatersrand Gold Mines.

4.6. — Refuse can be classified as domestic refuse and as trade or industrial wastes. Trade wastes are taken care of in large industrial areas by special removals where technical staff is employed to deal with such matters. These wastes are not extensive and should not occur as a major problem on the subcontinent. Domestic refuse is only collected and disposed of in the larger urban centres. In rural African towns, refuse disposal is not considered a problem by the inhabitants, refuse is merely dumped outside the individual's door and accumulates in the thoroughfare, attracting flies and rats. The collection and disposal of refuse from urban centres with mixed populations is a different matter. This refuse contains waste foods and discarded domestic articles; the bulk depending on what type of fuel is burnt in the kitchen stoves. Progress can be reported from towns such as Johannesburg and some other large cities situated mostly in the southern areas of the subcontinent, where storage takes place in steel bins: collection in specialised vehicle; and disposal usually by controlled tipping underdeal circumstances. These highly organised services can be compared with the best in the world.

4.7. — Waste water, from bathrooms and kitchens becomes a problem in heavily built up towns. Where sewerage systems exist waste water is passed into these, otherwise disposal is by conservancy on the premises and removal by tankers; or by subsoil drainage. The two latter methods are nearly always accompanied by a health hazard. In a subcontinent, one third of which is arid or semiarid, the disposal of waste-water should not be a great problem.

5. HOUSING AND TOWN PLANNING

5.1. — In no other branch of environmental sanitation of the problems of population pressures, rapid urbanisation, and a too-rapid economic expansion become more evident than in town planning and housing. The traditional haphazard conglomeration of huts forming an African village become a festering sore of insanitary practice, when transferred from their rural setting to the industrial areas. This state of affairs has in fact taken place in most countries on the subcontinent. Those states in the throes of industrial expansion being affected in greater proportion to the rapidity of the rate of such expansion.

5.1.1. — Rapid urbanisation of the indigenous African is taking place all over the continent. In most countries the urban populations have doubled or even trebled themselves in the post-war period, examples are: 1) Brazzaville population increased from 45,000 in 1940 to 79,000 in 1950; 2) Dakar population increased from 95,000 in 1940 to 225,000 in 1950.

5.2. — African communities living under original conditions and engaged in rural occupations are often referred to as living in «Villages». This; despite the fact that some of these so called «Villages» house 75,000 inhabitants or more. These communities live under conditions which vary only as to size and therefore only in the magnitude of their sanitary problems.

5.3. — Valuable progress has been made by introducing sanitary practises into indigenous building construction in schemes like the Anchou Resettlement Scheme in Nigeria. The Anchou Resettlement scheme is an outstanding example of how a whole community can be moed and established under sanitary conditions. On the other hand where housing conditions deteriorate or become inadequate in the industrialised areas there has been an increase in what might be called the social diseases. In fact tuberculosis, veneral disease, and other contagious diseases mainly respiratory have increased amongst the urbanised African throughout the subcontinent.

5.4. — In the Union of South Africa a total of £ 90,984,215 (273,000,000$) has been authorised to date from Government funds for housing of all categories. In addition Building Societies financed by privately subscribed shares have issued loan capital for building dwelling houses on an economic scale. Of the government funds already authorised £ 74,512,281 has actually been issued. The total new houses completed, under construction or approved for 1954-1955 was just under 9,600, about two-thirds of this total being for Africans. In addition money for subsidised housing has recently been made available from other sources. The Transvaal and Orange Free State Chamber of Mines has loaned £ 3,000,000 (9,000,000$) interest free to the Johannesburg Municipality for this purposes. Another expediency to alleviate insanitary conditions developing in shanty towns which are erected by squatters is the «Site and Service Plan» which has contributed not a little towards solving the actual housing shortage. A total of £ 11,000,000 (33,000,000$) has thus been spent on housing of all types. About 6.5 millions francs have been spent in the post-war period by France on urban and rural housing in her African dominions. The rise in urban populations in these areas has been extremely rapid, practically doubling the population of the larger towns within the last decade. Town-planning legislation and staff has been increased, although rural housing is still in a backward way. Villages consisting of the grouping of small single room houses, widely scattered inside a fenced to in area. This construction is flimsy with scant protection against the weather and are a high fire risk. There is a complete absence of sanitary arrangements.

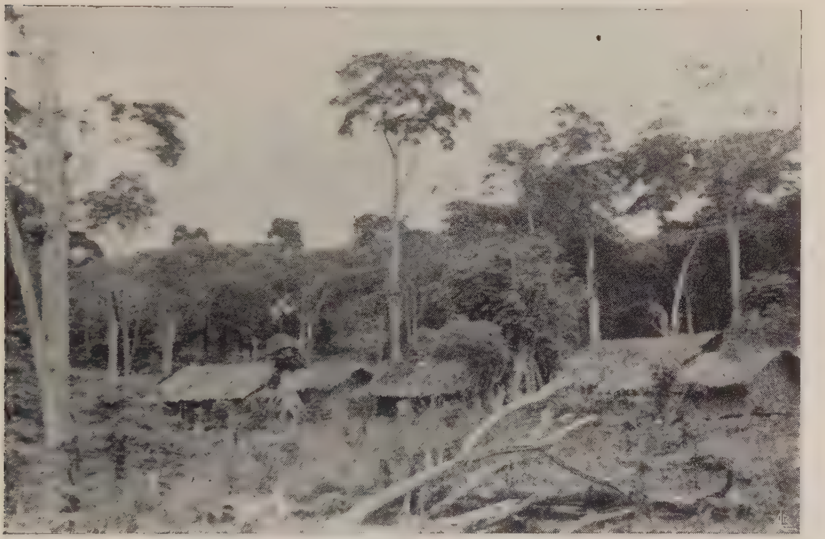

Fig. 7 — Clearing jungle for the erection of indigenous housing

(Photo H. J. Heinz)

In Uganda new building activity in urban areas has slowed down, while control of building standards in rural areas is most difficult. The Central African Federation has progressed in housing and town planning in the nine major urban centres but progress in the other urban or in rural housing is very slow. Much of the housing in Nigeria is inadequate, both in urban and rural areas, but town planning committees and further staff have been appointed in some of the major urban areas in order to expedite matters.

Fig. 8 — Indigenous housing in dry south-western Africa

(Photo Schulz).

5.5. — Slum conditions remain most obvious in the rapidly expanding industrial areas, chiefly in the southern part of the subcontinent where desperate efforts to catch up with housing requirements have been made. However, these efforts could only overtake the demand for adequate housing if colossal finance and an army of trained personnel were allocated only to this one aspect of environmental sanitation.

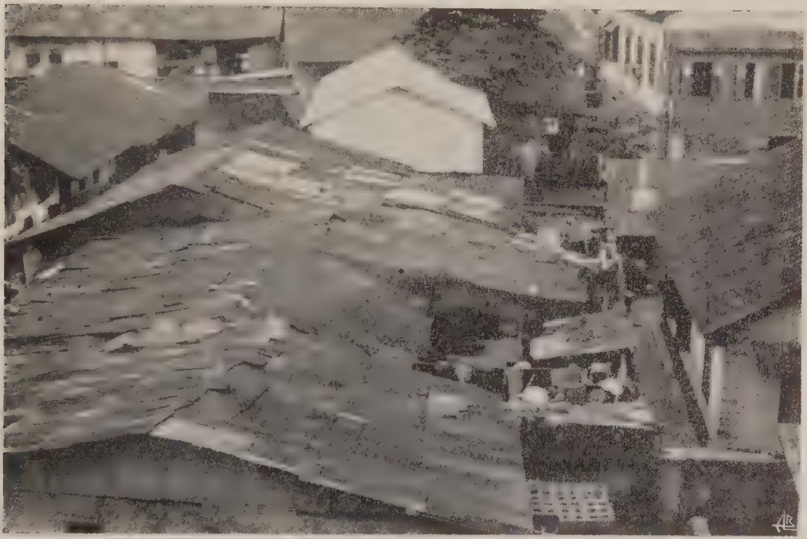

Fig. 9 — Aerial view of slum due to rapid urbanisation

(Photo H. J. Heinz)

5.6. — The social diseases i.e. those whose spread can also be attributed to bad housing and overcrowding, are becoming increasingly important as their prevalence is discovered by increased and improved diagnostic services. These diseases are widespread over the subcontinent. Consideration of the returns of the incidence of only one of these social diseases i.e., of tuberculosis, shows that it is mostly impossible to analyse even where these exist: a few well known generalities are quoted to show how bad the continental situation is. Uganda has 55 treatment villages besides mission facilites for the isolation and treatment of between 25-30,000 patients. Tanganika reports over 8,000 tuberculosis admissions yearly to hospitals. In the Union of South Africa facilities for the treatment of discovered cases of tuberculosis do not even approach the demand from the most privileged section of the population. Treatment is given in governmental hospitals while a semiprivate organisation known as Santa provides settlements for the treatment and holding of semicured cases pending their return to work. While tuberculosis seems to be on the

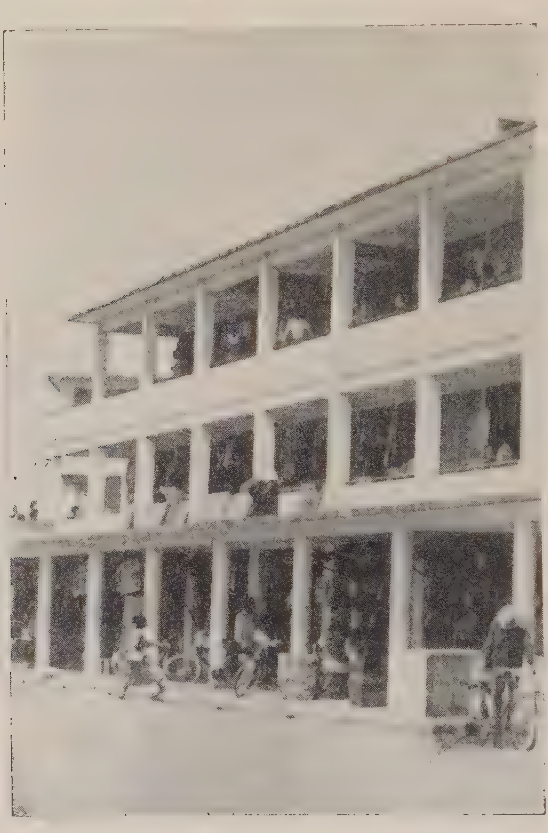

Fig. 10 — Good construction practice, but overcrowded due to influx into urban area

(Photo Schulz)

wayout on some of the other continents, it seems to be on the increase on the African continent, and we are merely moving on the fringe of one of the causal conditions.

6. CONTROL OF VECTOR-BORNE DISEASES

6.1. — The days of the great African epidemics seem to be over, partly because of the improved techniques of epidemic control such as 1) immunisation and 2) efficient vector-control by the use of the modern insecticides and partly due to other little known factors. Most certainly there has been a spontaneous remission in incidence as well as the virulance of many of the epidemic diseases which ravaged most of the subcontinent in the past. The incidence of Yellow Fever and Plague has certainly decreased beyond the limits which could be expected from the methods used for their control.

6.1.1. — Great progress can be claimed, however, in the control of many endemic vector-borne diseases. In the British territories of East and West Africa alone many specialised units are active on control and research on vector-borne diseases, while similar research and control are being actively carried out in the French African territories, the Belgian Congo, the Portuguese territories, the Central African Federation, and the Union of South Africa. Other states are carrying out control and research as a normal routine of their medical departments. International agencies such as WHO and the Rockefeller Foundation have co-operated to strengthen organisations and to aid research when requested to do so by the African States.

6.2. — *Yellow fever*. Lord Haily in «An African Survey» writes as follows «In spite of the discovery of systems of prevention and treatment, yellow fever remains a great potential danger to Africa» yet this seems to be one of the first of the great African epidemic diseases which seems to be retreating rapidly. Single cases appearing in any region are enough to excite great medical interest. Only a few cases, some of which are not verified are reported annually from yellow fever areas; although immunity to the mouse tests are continually being discovered, sometimes in embarrassing places.

6.2.1. — The year 1940 saw the worst yellow fever epidemic of modern times when 15,000 cases occurred in the Sudan, with a

10 % mortality. In 1951 another outbreak occurred in the eastern region of Nigeria when 5,500 cases occurred with 600 deaths. The French have innoculated nearly 4,000,000 persons from 1939-1954, and the *stegomya* index has been reduced in many places by the use of D.D.T. There is less than one case notified per annum in all the French African territories. West Nigeria reported 2 suspected cases for to year 1956: while one fatal case was reported from Sierre Leone where 13,200 vaccinations were carried out in one area only. In Ghana two suspected cases occurred, one being fatal which was coupled with an outbreak of jaundice: a survey is proceeding here to determine the extent of the infection.

6.2.2. — Although mosquito control measures are carried out as part of the routine public health duties by all African countries where mosquito-borne diseases may occur, the amount of work done is sufficient to protect only a small proportion of the population at risk, and is nearly always confined to the urban areas.

6.3. — *Plague.* In spite of the fact that one of the world's biggest enzootic plague areas exists in the southern part of the subcontinent while several smaller foci occur further north the incidence of human plague on the whole continent is rapidly decreasing. This regression in the incidence of human cases is in accordance with findings elsewhere in the world where these enzootic foci are to be found. It seems that we are seeing the receeding of the present plague pandemic which commenced more than 300 years ago.

6.3.1. — Progress can be claimed in limiting outbreaks which occur in rural areas, and in keeping plague away from the big towns by active antirodent and antiparasitic measures, particularly in the southern part of the subcontinent, where positive plague can always be picked up either in the primary rodent reservoir *(Tatera schintzi)* or in the flea vectors *(Phyloxera)* over vast areas in the Karroo and the Kalahari. Rodent destruction by poisoning and vector control by means of D.D.T. 10 % dust is continuously carried out where the plague surveillance service shows any possibilities of human cases occurring.

6.3.2. — Rodent control is passing from pure public health practice where methods of control where pioneered into other fields as the economic importance of rodent damage becomes more apparent. In French Africa active antiplague measures go on apace. Several hundreds of thousands of rats are killed in towns annually, while D.D.T. is used against flea vectors. More than 3,000,000 people were vaccinated against plague in between the years 1938-1951, although this measure now appears to have little value. Whereas 24,823 plague cases were treated in the years 1929-1938 this figure dropped to 4,587 during the years 1939-1951. Uganda reports no cases in recent years, but there are still enzootic areas in West Uganda and on the Congo borders, which have to be watched. Active antirodent measures are carried out in Swaziland although no cases have occurred for several years. Cases of plague were reported from the Congo in 1954 with 22 deaths. Sierre Leone carries out active antirodent measures in the form of trapping and poisoning although trapping has never been found practicable on a large enough scale. No cases have been reported from Bechuanaland recently. In the Central African Federation two outbreaks occurred recently in Northern Rhodesia, with 39 cases and 8 deaths; while no case has been reported from Southern Rhodesia for the last 20 years. Ghana reports 106,962 rats trapped in urban areas, but no reference is made to the possibility of further outbreaks of human plague. In Tanganyika plague remains an ever present threat, two outbreaks occurred recently after two silent years, in which D.D.T. was used for the control of the vectors. In 1952 there were 573 human cases with 100 deaths; 1953, 12 cases with 3 deaths; and 1956, 5 cases with 1 death. The number of outbreaks as well as the total number of cases has decreased in the Union of South Africa within the last two decades. This decrease in human plague is taking place even though smouldering plague can always be demonstrated in the wild rodent reservoir and its parasites over nearly a quarter of a million square miles of enzootic plague areas in the Karroo and the Kalahari desert, where a periodicity of 5-7 years can be predicted in between peak rodent population levels. A good plague surveillance and research service has been developed by the Union Health Department in co-operation with the South African Institute for Medical Research, which is also of great value to the neighbouring States.

6.4. — *Trypanosomiasis.* Despite wide areas under tsetse fly in West and East Africa, as well as Central and the subtropical South, few human cases are now reported from any of these areas. In the past cases increased when communications were first opened up in new fly areas; now fear is expressed again that with increased riverine traffic on the Volta and Kariba schemes this situation may occur again.

6.4.1. — Severe epidemics occurred at the turn of the century, particularly along the shores of the great African lakes, but since then tsetse fly control has made tremendous progress, particularly in the southern part of the subcontinent, where eradication is claimed of the vector of nagana from large tracts of subtropical South Africa by the use of aerial spraying of residual insecticides. In other areas fly control has been attempted by naturalistic means such as removal of riverine shade and the slaughter of all game present (a dubious precaution). The slaughter of game for this purpose seems not to be justified as the advantages have not been proved. Progress in the control of the disease has also been made by the removal of whole communities from fly areas and their resettlement in fly-free and more healthy environments. The Anchou Resettlement scheme carried out in Nigeria was amongst the first really progressive schemes of this nature, and proved conclusively the advantages which could be obtained in general public health and economic fields from such a resettlement of a local community. In French Africa 50 million people were examined for sleeping sickness up to the year 1954 and 8 ½ million injections of vaccine given to those requiring treatment. New cases reported from here dropped from 44,661 in 1929 to 16,751 in 1951. Uganda reports a small number of deaths from this disease which is still present in endemic form. Control measures carried out consists of resettlement of communities from infected parts in fly-free areas and spraying with residual insecticides. In Uganda the annual infectivity rates have been dropping steadily: 78 cases with 6 deaths reported for 1950; 38 cases with 2 deaths for 1952; and an increased rate of 108 cases with 1 death for 1956. Recently no verified cases have been reported from West Nigeria. The problem of control is becoming increasingly important in Liberia. Ten cases were reported from the N.W. of Bechuanaland where the distribution is strictly limited. In the Central African Federation large areas are fly-infested but few human cases accur; the swing-fog insecticidal applicator is used here for fly-control. A spontaneous reduction in the incidence of disease is taking place in Ghana, where 3 cases occurred in Togoland according to the most recent report. It is feared, however, that the incidence may rise again with the increased riverine traffic due to the completion of the Volta Dam Project. On the other hand the incidence is increasing in Tanganyika from 346 cases in 1952; 736 cases in 1953; 1230 cases in 1954; 923 cases in 1955; 646 cases in 1956. Resettlement is considered the answer to the problem in Nigeria and other parts of the west coast where many cases still occur annually. Some progress in the control of the fly in infested areas and treatment of discovered cases can be reported from the Portuguese territories as well as from the Belgian Congo. Consideration of the data given above although vague shows what progress has been made, but also emphasises how much is still left undone.

6.5. — *Malaria.* The intense endemicity African malaria coupled with the great efficiency of the vectors has made the malaria control problem in Africa a very difficult one; which has posed special problems which have not been encountered elsewhere. Pampana considers that there are about 116,000,000 persons at risk in the malarious areas of Africa. Only those areas which are free from malaria in Africa are those unsuitable for the propagation of its vectors, such as the dry desert and semidesert areas and some highlands, and even in those places epidemics can sometimes be expected. It is only in the extreme temperate South that malaria does not occur. The Malaria Conference held at Kampala under the auspices of WHO and CCTA in 1950 considered and recognised the difficulties in the way of large scale malaria control programs in Africa. In spite of the problems set by efficient and abundant vectors, by great distances, by the type of building materials used, great progress can be reported by all states.

6.5.1. — Pilot schemes started by the French in 1952-3 in Senegal, Haute Volta, Togoland, Dahomy and the French Cameroons have been expanded to protect all urban areas exceeding 2,000 inhabitants. By 1955 about one million people were protected. D.D.T. is sprayed by mobile sanitation groups. There is no new transmission in some of these areas and the mortality rate from malaria has fallen from first importance as a cause of death to 5th. In Uganda malaria is still hyperendemic in many parts, and it is still considered a very serious public health threat. Cases treated in 1954 numbered 97,526 with 201 deaths, although cases amongst expatriates has decreased enormously in the same time due to control measures. Swaziland reports a decrease in cases from 807 in 1950 to 105 in 1955: the number of huts sprayed with BHC in that year totalled 10,276: malaria rates now vary from 0.7 % in the dryer Bushveld to 3.1 % in the irrigated areas. One vector — *A. funestus* — seems to have completely disappeared from Swaziland due to the control measures carried out, while *A. gambiae*, although still prevalent, is rarely found in adult form in huts. Total interruption of transmission for two years has been achieved in one middle-veld area where the control program has been been replaced with a surveilance service. In 1956 malaria in West Nigeria was still the commonest single disease for which treatment is sought: hospital admissions and treatments for this disease being 67,126. Mosquito control is carried out by swamp reclamation, larvicidal work and a small amount of adulticidal work is carried out with Paris Green and BHC. In 1954 a total of 120,000 persons were protected against malaria in part of the Sokoto Province, since then this protection has been expanded to take in the whole Province. It was found here that some *Anopheles* species seemed resistant to both Dieldrin and BHC. A few years ago the problem in Liberia was most serious with about 30 % of the population infected and black water fever was prevalent. A pilot project was commenced in 1953 for the control of malaria which initially protected 54,000 people. This project was enlarged later to protect 250,000. The Belgian Congo has shown remarkable progress in the control of malaria with residual insecticides, malaria has been practically eradicated from the large industrial and mining centres. Sierre Leone reports quarterly residual spraying of 20,000 huts in one town alone, and although there were 21,500 hospital admissions for malaria for the year it is considered that the incidence is receeding. Bechuanaland although having more favourable conditions in most parts for the control and eradication of malaria reported 1962 cases for 1956. A more intensive control policy has now been set for the future. Barrier spraying has been carried out round 45 African centres in the Central African Federation, in 21,000 square miles thus protecting a population of 2,000,000. During the 1955-6 season 280,775 habitats were sprayed in Southern Rhodesia; 7,122 huts in Northern Rhodesia, and 43,146 habitats in Nyasaland. Transmission of malaria has been interrupted on the Copper-Belt for several years by the intensive malaria control practiced there. A nine year program in which BHC 10 % wettable powder is used has served to protect most of the population. In Ghana small progress can be reported due to lack of finances and of trained personnel. Most control seems to be done by oiling water surfaces in the vicinity of the larger towns. The Tanganyika malaria pattern remains constant, with a steady improvement in the urban centres: total hospital admissions for all forms of malaria were 119,026 for 1956. The East African Institute of Malaria and Vector-Borne Diseases, which is operative throughout the territory, is conducting research into the efficiency of residual house spray, the life history of *A. gambiae,* and is supervising control schemes at Uganda, Zanzibar and in the Pari-Taveta area where 100,000 persons are protected. The Union of South Africa has carried out extensive malaria control programs in the Transvaal and Natal, where approximately 1,000,000 inhabitants living in about 60,000 square miles are protected. While the situation approaches complete control, interuption of malarial transmission for several consequetive years has not yet been achieved. Ironically enough the conquest of malaria in the Transvaal has led to an extension of the areas in which bilharzia transmition can now be expected, by making farming by irrigation possible of land which has been left alone for centuries.

6.5.2. — Greater success on the subcontinent is seriously hampered by the lack of inter-state co-operation, and the failure of governments to expand existing control programs into malaria eradication projects. In spite of research proceeding at various centres we are not yet able to say whether the vectors behave the same over the subcontinent and if insecticidal resistance, behaviouristic or biological, is a fact or not. Neither is it possible for any single state to formulate a policy for the complete eradication of transmission and for the economic maintenance of such a condition when this will become a fact. Of 116,000,000 people at risk from malaria in Africa South of the Sahara, Pampana considers only 18,000,000 are today being adequately protected.

6.5. — *Relapsing Fevers, Tick and Louse Types.* Louse-borne relapsing fever has a sporadic distribution throughout the subcontinent and the incidence of cases is influenced by the seasons. It appears regularly in parts of Western and Southern Africa. Tick-borne relapsing fever is present throughout the subcontinent

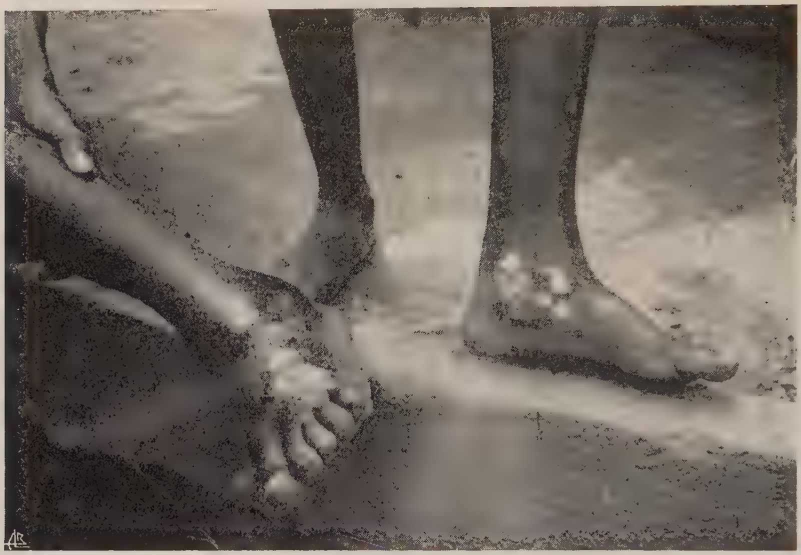

Fig. 11 — More crippling guinea-worm lesions

(Photo H. J. Heinz)

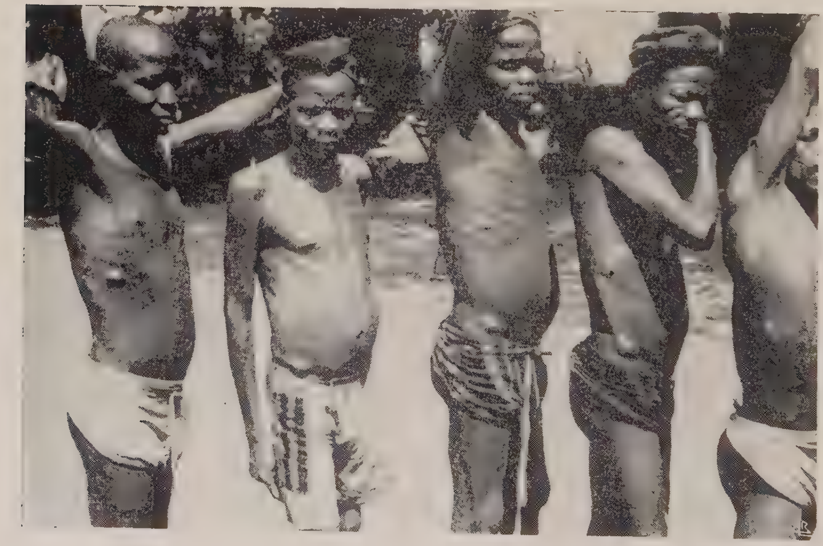

Fig. 12 — Nodules of onchocerciasis

(Photo H. J. Heinz)

and depends for its transmission on the distribution of the vector *(O. moubata)*. The spread of the latter type depends much on the movement of migratory labourers and their use of tick-infested rest houses along the migratory routes. The use of residual insecticides for delousing people gave great promise for the elimination of louse-borne diseases, but we are now confronted with the possibility of large scale resistance in lice. This resistance of lice to the residual insecticides has already been confirmed in the Transkei Native Reserve of the Union of South Africa.

6.5.3. — Progress in the elimination of the tick-borne type can be reported from Uganda where it is present mostly in the migrant labourers. A total of 28 cases have been reported, the decrease is due to natural causes as well as the use of D.D.T. dust and a higher standard of cleanliness in rest houses. The Central African Federation reports a reduction in cases from 3403 with 41 deaths in 1953 to 2744 cases with 30 deaths in 1954. Further progress on the subcontinent is difficult to show in the absence of information dealing with the incidence of the disease in other States of Africa.

6.6. — The importance of onchocerciasis was fully appreciated at the Conference held at Léopoldville in October 1954. Its distribution is throughout Tropical Africa.

6.6.1. — Surveys carried out in the Belgian Congo place the infected rate of the population as high as 88 % in villages close to rivers and streams. Uganda is becoming increasingly aware of the infection; in 1952 a total of 21 cases was reported, whereas in 1956 this had increased to 661 cases. Control of *Simulium* is attempted with D.D.T. in several areas, while a claim is made that the vector has been completely eradicated from a considerable lenght of the Nile. A survey of the distribution of the vector is being carried out in Sierre Leone. The incidence of infection is high in Ghana. Adequate control of *Simulium* has been claimed in important areas in Kenya, Uganda and the Belgian Congo. However, the incidence of infection in the African population has been shown in surveys to be disturbingly high, over the whole subcontinent.

6.7. — *Dracontiasis*. This disease is present throughout the wet tropics, with a high incidence reported in West Africa where rates vary from 16 % in Nigeria to 23.5 % in Portuguese Guinea. The infectivity rates have been greatly decreased in Uganda in the last

25 years. This is the most crippling of all endemic diseases in Ghana where its distribution has been shown to be intimately connected with the method of obtaining rural water supplies.

6.8. — *Bilharziasis.* Little reliable information can be reported either on the extent to which the problem of bilharzia is present or to what extent African States are already combatting this disease; a general statement only can be made to the effect that bilharzia is widespread and endemic in Africa South of the Sahara. Its spread is not only being accelerated by the increasing use of irrigation, but more importance is being attached to the discovery of new cases as the economy of backward areas improves and diagnostic services are improved. Uganda reports that its prevalence is quite unkown. The East African Institute of Malaria and Vector-Borne Diseases carries out ecological studies and makes surveys: some reduction in the incidence has been brought about by the use of molluscicides, although the problem of control still remains an enormous one. In West Nigeria the disease is endemic. In the Congo there were 51,259 hospital admission in 1954. The Central African Federation reports wide-spread endemicity. Control work with copper salts on a large scale has been carried out there. The WHO snail identification service is located in Southern Rhodesia. Surveys are proceeding in Ghana, to determine the incidence and distribution, but bilharzia is not considered at present as important as the incidence of other fluke and filararial diseases. Bilharzia occurs extensively in Tanganyika but exact information is lacking, consequently a request has been made to WHO to carry out surveys to determine the incidence and distribution. It is thought that the incidence is increasing due to the progress made in dam building throughout the country, hospital admissions for 1956 were 3092. In the Union of South Africa some progress has been made in research on the taxonomy and ecology of the vectors; while control measures have also been started over a wide area by the anti-malarial organisation.

6.9. — *Other filarial infections* (elephantiasis, loa-loa and leishmaniasis). Some of these infections are most common throughout West and East Africa, especially along the coast and round the great lakes. Some surveys in these places show that as many as 40% of the local populations are infected with loa-loa.

Leishmaniasis is not common, although it is now also reported from French West Africa. The infection may have been introduced by troop movements during the recent world war. No detailed information is available on the plans made for its control in the areas affected.

6.10. — *Musca Domestica*. Reports showing what progress has been made on the control of domestic flies over the subcontinent is most difficult to obtain. A good control of the environment usually ensures a good measure of fly control as well. Recently where this could not be obtained fast enough reliance was placed on the special use of residual insecticides to bring this about. However, widespread reports are now made on the increasing resistances of domestic flies to the carbonated hydrocarbons and related substances. Amongst the places from where resistance is definitely reported is the Union of South Africa and Swaziland.

6.10.1. — Not only can the density of flies be used as an index for judging the standard of sanitation progress achieved in an area, but this density can also be related to the incidence of such diseases which are commonly spread by flies. If we apply this measure of progress of onvironmental sanitation to the subcontinent, then the real lack of progress becomes painfully apparent when judged by the swarms of flies found everywhere in association with the high incidence of fly-borne diseases prevailent in Africa South of the Sahara. *Musca Sorbens* is assumming an increasing public health importance as its association with the spread of trachoma becomes more clearly established. This crippling eye disease is endemic throughout the subcontinent, although no exact information is available. Several surveys have been made in the Native Reserves of the Union of South Africa and the extent to which various degrees of blindness is due to this disease has been found most disturbing.

7. MEDICAL POLICY — GENERAL TRENDS

Medical policy generally in Africa South of the Sahara has grown out of early governmental assistance given to Christian Medical Missions established to treat the more acute diseases. Hailey gives the first record of a missionary hospital erected on the subcontinent for the treatment of sick indigenes as being in the year 1518 when a religious order erected a hospital in Moçambique. In 1810 Doctor Van Der Kamp, a medical missionary in South Africa completed a considerable book on midwifery for the benefit of the Hottentots. Lovedale College was founded in the Eastern Cape by the Scottish Presbetarian Mission in 1841 and Victoria Hospital added to it for the treatment of the sick and the training of African nurses shortly after. These first curative medical services established by mission enterprises were followed by a constant expansion of medical activities until to-day there is an important net-work of missionary hospitals over the subcontinent.

7.1. — Governments have in the past found it convenient to subsidise these institutions, in addition to supplying a small state service. A further stage was reached when medical policy demanded an expansion in the form of a preventive medical service. Usually this was grafted on to the existing service and was subordinate both in numbers of personnel and in budget and hence second also in the field of activities.

7.2. — This situation has persisted to the present day, when few African States can show environmental sanitation (preventive medical services) activities comparable to or exceeding that of the curative medical service. An estimation places the total number of hospital beds on the subcontinent at approximately 225,000 in various types of institutions (with necessary medical and nursing staffs): while it is considered that there are no more than 3,500 public health workers of all categories actively employed in the same areas. Hospital admissions (in-and-out patients) in one African State alone for a single preventable disease such as malaria for 1956 was 119,013. This means that about 10 % of the total population passed through the portals of the hospitals for that year. In another State more than 100,000 persons received treatment for a condition which could have been prevented by improving water supplies. Again in another State more than half a million in-and-out patients were treated in the same year for all infective and parasitic diseases: while in yet another State the total patients attending at dispensaries and hospitals increased from 546,346 in 1952-3 to 713,719 in 1953-4. Much of this increase could have been prevented by the efficient operation of an expanded preventive medical service in the States concerned.

7.3. — It is possible to obtain impressive results in public health, especially with epidemic control, by the use of inexpensive mass proceedures such as innoculation against specific disease or by the spraying of residual insecticides on a large scale for the control of insect-borne diseases by small numbers of expert personnel when merely passive support is required of the public. But to obtain permanent gains from such campaigns it is necessary to base all preventive health operations on indigenous agencies staffed with adequately trained personnel in sufficient numbers sincerely convinced of the merits of the system which they are applying.

8. SUMMARY AND DISCUSSION

An assessment of the progress of environmental sanitation in Africa South of the Sahara cannot be attempted without consideration of other associated factors such as the geographic and climatic conditions forming the habitat, the socio-economic trends newly awakened, and the demographic trends resulting directly from these.

8.1. — Africa South of the Sahara consists of almost 20 % of the world's land surface inhabited by about 7 % of the world's population. The economic standard considered on a per population basis is one of the lowest in the world. Africa in 1949 accounted for only 8 % of the world's total imports and 5,9 % of total exports, the income level per capital varies between $50 (£ 18-10-00) for the tropical central and northern sectors and $ 250 (£ 80-00-00) for the more temperate southern sectors. Income obtained from mining, building, transport and general industry is increasing in all countries except the Gold Coast, Nigeria and Uganda. In these latter countries subsistence agriculture is still the most importance occupation. The capital investment required for the economic development of the subcontinent would be about $ 8,000 million (£ 3000 mil) spread over ten years: almost all risk capital would have to be foreign. Expenditure for public health and social services can be considered extra to this figure.

8.2. — It is a fact that most technical direction in Africa required is to-day being provided by the citizens of countries outside Africa. While African participation in advancement is increasing, this will have to be at a greater rate if further progress is to be made.

8.3. — Density of population is related to climate, varying from 4.6 to 6.7 per square mile. Population is mostly indigenous with Bantu predominating in the east and southern areas, where also most of the European populations is concentrated.

8.4. — Human fertility remains at an extremely high level, while mortality rates, especially infantile mortality rates, are being lowered all over the subcontinent. This release of the natural brake on population levels is one of the most important aspects of the progress of the science of environmental sanitation, and contains a challenge to all future planning, social as well as public health.

8.5. — Safe water supplies are reticulated to most of the big industrial urban centres and progress is being continued in most countries where money is available, with large sums being devoted to new works and extension of old ones (Union of South Africa, £ 99,762,779 for 1955; France, 5 billion francs). The State of rural water supplies remains shockingly inadequate even to large rural towns. Water in these areas is still obtained as it was centuries ago from mud pools or the banks of rivers, or on the road side. In many cases it is carried by hand for miles. The incidence of water-borne diseases is increasing over the subcontinent.

8.6. — The problem of human wastes disposal in an appalling one, even in large industrialised urban communities, while in rural areas no pretense is even made to meet this problem. Provision of water-borne sewage systems to towns is limited by the overall shortage of water. The major urban centres nearly all have partial water-borne sewage, coupled in most cases with a bucket conservancy system. Disposal of faeces varies from modern sewage digestion to crude broad irrigation or flooding. Only in the south is some attempt made to compost crude sewage. In large and small rural communities the domestic animals still have to do their share in faeces disposal in a closed economy. Refuse is collected and disposed of under conditions varying from the most primitive (dumping outside the door into thoroughfare) to those which can be compared to the best in the world (storage in steel containers; collected by specialised vehicle; and disposed of by controlled tipping). Waste water is no great problem in a continent which contains some of the greatest deserts in the world and where relative humidity is consequently low.

8.6.1. — The faecal diseases — flat and round worm infections, amoebiasis, dysentery and typhoid, are widespread on the Sub-continent and especially so in rural areas. There is no indication of a decrease in these infections.

8.7. — Housing and town planning can show progress only in relation to past efforts, but cannot keep pace with African Fertility combined with the rapid urbanisation taking place as a result of the expanding economy all over the continent. Astronomical finance figures would be required to advance the progress to keep pace with the demand. In the undisturbed rural areas the African still looks after his own housing and uses materials which come easily to hand. Some little progress has been made in teaching the African to graft good housing procedures on to his traditional ways. The social diseases connected with slum conditions, such as T.B., V.D. and bronchial diseases, are becoming increasingly important as diagnostic services increase with the expanding industrialisation.

8.8. — In one great field can the science of environmental sanitation claim amazing results — that of the control of vector-borne diseases. While spontaneous remission of the great epidemic diseases of Africa is no doubt also taking place, great credit can be claimed in the progress made in yellow controlling vector-borne diseases in many countries: yellow-fever control in West and Central Africa: control of malaria in the Union of South Africa and parts of East and West Africa, and its elimination on the Copper Belt in Northern Rhodesia: diminishing outbreaks of plague in Southern and Central Africa: control and almost elimination of sleeping sickness in the North, Central and Eastern endemic areas. — The incidence of all vector-borne disease has been further reduced by specialised control of the environment. Much remains to be done, however, especially as far as some filarial infections as well as bilharzia, are concerned.

8.9. — Progress is hampered not only by lack of trained personnel but also by material from which to recruit trainees: by lack of adequate budgets: and also by the tradition of expanding only curative medical services. This is an aspect of policy which has grown out of the provision in the past to medical missionaries. An estimate places the total number of hospital beds available at about 250,000 in all types of institutions (1 bed per 800 inhabitants), while there are not more than 3,500 public health workers of all categories actively employed on the subcontinent (1 worker per 60,000 inhabitants). Small public health staffs, well trained and well directed, can obtain considerable results especially in epidemic disease control where little more than passive support is required from the general public. However, permanent gains in the field of public health cannot be established without active participation of the whole community.

8.10. — In Africa the further progress of the successful control of the environment must be accompanied by social progress as well as by an expanding economic position over the whole subcontinent, otherwise the present low levels will be further depressed. The progress of public health and environmental sanitation must take into account the problems arising from decreasing mortality which they bring about and which are rapidly affecting the economics of the African family.

8.11. — Any policy of outside assistance should be based on helping the African to help himself, and public health planning should be integrated with a master plan to develop education, agriculture, communications and industry at the same time.

ACKNOWLEDGEMENT

I am greatly indebted to Professor E. H. Cluver, Director of the S.A.I.M.R., for allowing me to use the Library facilities for the compilation of this work, as well as providing typing facilities: he has shown great interest at all times in my work. Dr. Ordman, Chief of the Allergy Department, and Dr. Heinz, Chief of the Parasitology Department of the S.A.I.M.R., and Dr. Peacock, Lecturer in Public Health at the University of the Witwatersrand and M.O.H. of the City of Germiston, very kindly undertook to help me revise the manuscript.

The Director, Mr. Robertson, and the Librarian, Miss Saul, of the South African Institute for International Affairs, are thanked for their kind cooperation and for the use of the Library of that Institute.

I feel that any success attained in presenting the progress of Environmental Sanitation in Africa South of the Sahara is due to the interest shown and the assistance given to me by them.

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GENERAL DISCUSSION

Dr. Ernest Paulini (Brasil) — I'd like to comment the very interesting communication of Dr. Berti.

In an investigation made together with Dr. Ivan Ricciardi on the fly density and breeding in some areas af the Federal District of Brasil about four years ago, we found that domestic wastes represented the most frequent breeding places but poultry farms were the heaviest ones. We found also that it was not the animal fecal material but the food waste mixed with some spilled drinking water, which gave the heaviest breeding of fly larvae. The control of breeding was easily stopped in these special conditions by separating the food containers and those of drinking containers thus not permitting their mixing on the floor. We believe that where ever possible sanitation practice should be preferred in fly control since insecticides give only transitory control.

Ing. Arturo Berti (Venezuela) — El Ing. Berti respondió al Mayor Schulz que en Venezuela se ha investigado para distinguir entre *Musca domestica viccina* y *Musca domestica domestica*. Que los tres géneros *Musca, Sarcophagula* y *Drosophila* han desarrollado resistencia a los insecticidas clorados y que comenzan a mostrar alguna resistencia al *Diazinon*.

Mayor Schulz (U. South Africa) — *a*) Has any investigation been made to divide *Musca domestica* into subrace to demonstrate rational resistance to residual insecticides?

In South Africa we have too problems of *Musca sorbens* connected with blindness into Reserves.

b) In Africa we can confirm that water is n° 1 natural resource, particulary in arid areas.

An expensive failure in water conservation was Avis Dam in Wind Lack (S. W. A.) when it heavy burst in first year and remained empty for 5 years after.

Can any examples be given in the use of a closed circuit of urban water supplies?

Rural supplies can sometimes be done cheaply. The possibility of tube-wells should be explaited.

DR. DAVEY (U.S.A.) — I might point out an example in the U.S.A. which apprroaches a closed circuit. At the Grand Canyon National Park water is taken from the Colorado river and treated at the park site. The effluent from the Park sewage treatment plant reaches bacteriological standards for drinking water. However for esthetic reasons the water is used for irrigation purposes.

El Ing. Baity se excusó de responder en nombre del Prof. Steel pero hizo el siguiente comentario:

In many parts of the world, polluted water and sewage are being used extensively for agritultural irrigation and even for the recharge of underground water strata. It is reasonable to expect that such uses of sewage will increase.

2ND SESSION: *Thursday, September, 11th — 9:00 to 10:30 a.m. Room A*

The meeting was held under the Chairmanship of Br. T. H. Davey.

The Rapporteur was Ing. Arturo Luis Berti and the Secretary was Eng.° Salomão Q. Levy.

The following papers were presentend and discussed:

SALUD OCUPACIONAL EN VENEZUELA: ESTADO ACTUAL Y POSIBLE DESARROLLO

RAFAEL RÍSQUEZ-IRIBARREN [1]

El desarrollo de una especialidad médica o lo que tiene aún mayor trascendencia como es la creación o extensión de una actividad dentro de los planes de la salud pública es un complejo proceso en el cual entran en juego numerosos factores. Hace pocos meses tuvo ocasión de presentar ante la Academía Nacional de Medicina un trabajo en el cual estudiaba las bases y las posibles actividades de un programa de salud ocupacional (1). Este trabajo es un complemento de aquel, pues aquí me refiero directamente a los factores que han jugado y juegan papel determinante para acelerar o atrasar su desarrollo, o que no pueden ser apartados, sino por el contrario incorporados dentro de una adecuada planificación. Trataremos de analizar algunos de éllos a fin de darnos una idea de lo que está sucediendo en Venezuela (desde comienzos del siglo.

La legislación laboral moderna tiene sus comienzos en los códigos de minas, el primero de los cuales aparece el año 1909. Su evo-

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[1] Presidente del Consejo Directivo del Instituto Venezolano de los Seguros Sociales.

Proc. 6th Int. Congr. Trop. Med. and Malar., 6: 844-856, 1959

lución es lenta; un proyecto específico de legislación del trabajo fué rechazado por el Congreso en 1916, y la primera ley especializada aparece en 1936.

En lo referente a salud ocupacional y a partir de 1936 la legislación laboral venezolana de manera directa ha influido para garantizar los derechos de los trabajadores en los casos de accidentes y enfermedades profesionales y la provisión de permisos materiales. Ha servido de manera indirecta para el establecimiento de contratos colectivos que mejoran las prestaciones sociales establecidas por la Ley, considerados sus prescripciones como un mínimo obligatorio. Desgraciadamente y por circumstancias históricas lamentables el desarrollo sindical fué interrumpido en los últimos diez años, evitando asi un armonioso desarrollo de actividades en beneficio de la salud de los trabajadores.

La labor desarrollada por el Ministerio del Trabajo como organismo encargado de la aplicación y vigilancia de las leyes no podía escapar a estas condiciones. Antes, y aún después de la instalación del Seguro Social, ha sido encargado de recoger los datos estadísticos sobre accidentes y enfermedades profesionales. Desde el punto de vista salud ocupacional su labor ha sido reducida a garantizar la prestación económica correspondiente; salvo muy escasa labor en campañas de prevención de accidentes.

El futuro del Ministerio del Trabajo en labores directas de salud ocupacional es muy reducido debido a la creación del Seguro Social y debería abocarse de inmediato a una revisión de los sistemas de recolección de estadísticas y definir si aparte de lo necesario para la vigilancia del cumplimiento legal no sería más conveniente que el complejo problema de accidentes pasar a estudio y control de otros organismos nacionales, como lo sería la División de Salud Ocupacional del Ministerio de Sanidad, en coordinación con el Seguro Social en las zonas donde éste se aplique.

El petróleo descubierto en grandes cantidades que ha llevado a Venezuela a ser el segundo país productor del mundo ha sido un gran factor en el desarrollo de las actividades de medicina ocupacional. Es interesante hacer notar que durante muchos años los servicios médicos de la industria vinieron a ser una necesidad en zonas alejadas de centros urbanos y no precisamente de salud ocupacional.

No debemos olvidar que escasamente hace menos de veinte años (1941) la población venezolana en concentraciones menores de mil habitantes constituía el 60.75 % del total.

Las empresas petroleras, con mayor o menos intensidad enfocaron como ha sido siempre lo tradicional un programa de prevención de accidentes y de lucha de saneamiento. El programa de saneamiento varió de acuerdo con el nivel de los empleados así como también él de asistencia médica, y respondía principalmente a la necesidad de luchar contra el paludismo que constituyó una de las mayores causas de morbosidad y mortalidad. El elevado costo de la lucha antipalúdica antes de los nuevos insecticidas limitó esta campaña a una zona de seguridad dentro de los campamentos de trabajadores extranjeros. Basta citar como hecho demostrativo que para 1946, en un importante sector, el coeficiente de morbosidad era de 300 por mil.

Terminada la segunda guerra mundial en algunas de las grandes empresas comenzaron a desarrollarse actividades de medicina preventiva y saneamiento mediante el empleo de espcialistas calificados nacionales y extranjeros.

Actualmente y de modo general se observa una progresiva transformación de las empresas petroleras a limitar sus programas a una medicina ocupacional del tipo que se usa en Estados Unidos del Norte: responsabilidad de asistencia por accidentes industriales y enfermedades profesionales; exámenos periódicos de salud y asistencia a afecciones de tipo ambulatorio. Como complemento a estas actividades, la utilización de los servicios del Seguro Social donde éstos existen y el desarrollo de seguros privados de hospitalización. Es interesante observar que llegan a la construcción de hospitales, que luego hacen manejar por contratistas extranjeros a fin de poder introducir los planos de seguros privados e irse independizando de la prestación de servicios médicos a familiares. De seguida nos referimos al Seguro Social, pero deseamos observar que los objetivos perseguidos por estas empresas son contrarios al espíritu que priva en el país, con tendencia a considerar la defensa de la salud como obligación estadal o combinación con las empresas y trabajadores. No hay frente a estos problemas, ni en los médicos, ni en la población, lo que podríamos llamar un terror al fantasma de socialización, pues el gremio médico acepta los servicios médicos del estado siempre que se le dé el derecho a contratación y se respeten entre otros principios básicos los de : relación médico-paciente, participación en organismos directivos y oportunidades de mantener un nivel científico adecuado mediante la preparación constante del personal y mantenimiento de un elevado nivel de las instalaciones.

Desgraciadamente diez años de dictadura implacable permitieron la creación de nuevas situaciones e impidieron el libre juego de opinión.

Las empresas petroleras en el campo de salud ocupacional tienen hechos positivos que pueden resumirse así: 1) antes de 1936, el haber construído y mantenido los mejores hospitales del país; 2) el haber servido de ejemplo para demostrar que en mayor o menor amplitud y de acuerdo con el número de trabajadores es económicamente ventajoso para la empresa mantener los servicios médicos que ordena la Ley; 3) el haber demostrado la necesidad y la ventaja económica al incorporar personal calificado como ingenieros sanitarios y de industria, entomólogos en labores preventivas; y 4) el haber otorgado oportunidad para estudios de post-grado a un grupo de profesionales; y 5) el haber servido de centro de preparación para administración médica general a la mayor parte de sus profesionales.

La labor actual de las empresas petroleras tiende como dijimos anteriormente a repetir los métodos de los Estados Unidos de América que consideramos restringidos y perjudiciales en el caso de nuestro país, ya que éllos corresponden a la situación económica y condiciones sociales de esa nación diferentes a los de Venezuela. El cambio ocurrido en el Gobierno permitirá tomar medidas orientadoras tanto para las empresas como para los trabajadores a fin de obtener un desarrollo cónsono con las necesidades del país.

La creación del Seguro Social en 1944 y su extensión progresiva ha sido un segundo factor en el desarrolllo de la salud ocupacional, sin mencionar por obvias el establecimiento del concepto de previsión y la participación del indivíduo para la defensa de su salud y otras características interesantes al propio sistema; la contribución del seguro hay que analizarla bajo dos aspectos: en las zonas no petroleras y en las zonas petroleras.

De modo general el Seguro Social ha tenido graves defectos desde el momento mismo de su instalación. Algunos por su condición misma de nuevo factor social que encontró, tanto a patronos, como a trabajadores, como a médicos, no preparados para establecer condiciones y carentes de experiencia para preveer muchos inconvenientes. Por otra parte, el Gobierno mismo careció al principio de suficiente visión para insistir en que se trataba de un nuevo elemento en el desarrollo de los programas de salud pública y que el hecho de tener como contribuyentes a patronos y trabajadores es la consecuencia de un principio de justicia social que obliga a cada quien a contribuir de acuerdo con sus posibilidades. El haber permitido que se explote el concepto de que existe una asistencia médica para el indigente y otra para el asegurado, cuando ambos son patrocinados por el Estado, ha tenido y tiene graves consecuencias.

En las zonas no petroleras, el Seguro Social mejoró la asistencia médica global de sus beneficiarios, pues, salvo muy contadas empresas animadas por el espíritu social de sus dirigentes, tenían servicios médicos. Esta mejora fué grande sobretodo desde el punto de vista económico, pues no sólo reducía los costos de asistencia médica sino permitía gozar de prestaciones económicas durante los períodos de incapacidad. Hay que pensar que al iniciarse el Seguro Social un reducido número de obreros estaba protegido contra accidentes y enfermedades comunes por contratos colectivos.

En las zonas petroleras la situación era y es totalmente distinta. Por legislación y contratos colectivos los obreros gozaban de los beneficios inmediatos del Seguro Social. La diferencia, como tuve ocasión de decir hace varios años (2), estaba en: 1) indemnizaciones en efectivo en vez de pensiones para los accidentes industriales, y 2) la falta de asistencia hospitalaria para los familiares que depende de las facilidades existentes en las empresas y la cual venía a ser practicamente inexistente para la mitad de éstos. Fuimos y seguimos siendo partidarios que lo que ha debido hacerse es estudiar fórmulas mediante prismas diferenciales y arreglos administrativos a fin de ampliar lo existente y no cambiar de manera total lo que existía y que históricamente tenía un desarrollo que no podía preveerse.

Hay un hecho negativo de importancia como es el impacto negativo a la economía nacional, al reducir los trabajadores y facilidades médicas de las empresas con motivo de las extensiones del Seguro Social. Es muy dificil demostrar que se han obtenido ventajas nacionales mediante estas extensiones.

La labor desarrollada por el Instituto Venezolano de los Seguros Sociales ha tenido énfasis en la prevención de accidentes y es muy poco lo realizado en cuanto a labor médica, bien por consultas, especialidades o por métodos de laboratorio.

Entre sus labores merece destacarse el establecimiento de un Servicio de Rehabilitación en el Hospital José María Vargas de La Guaira. El alcance en cuanto a volumen de trabajo no es grande en comparación con otras actividades, pero tiene como característica principal la haber sido establecido con personal técnico idóneo, mediante colaboración con la organización de las Naciones Unidas.

El problema que se plantea para el Instituto de los Seguros Sociales se semeja al nacional: carencia de personal técnico para la rápida ampliación de estas actividades. Ante dicho problema la única solución es la de coordinar actividades y evitar gastos en duplicaciones de servicios e instalaciones que deberían dedicarse a la preparación de personal.

La propia ley del Seguro Social, al no establecer estímulos para la prevención de accidentes y una clasificación a priori de los riesgos de las industrias sin tomar en cuenta sus coeficientes de accidentes industriales, es un factor negativo en la necesaria campaña de prevención de accidentes. No menos importante es la falta de asistencia médica preventiva, hecho que también repercute sobre la educación de trabajadores y patronos, además de los perjuicios económicos contra el Instituto y las lógicas repercusiones contra la salud de los trabajadores.

La modificación de la legislación vigente es indispensable y aunque sólo cubriera estos dos aspectos podría justificarse.

Los riesgos cubiertos por el Seguro Social son el de enfermedad-maternidad y de accidentes y enfermedades profesionales. Se ha extendido geográficamente a Caracas, Maracaibo, Valencia y Barquisimeto que son las cuatro primeras ciudades de la República y a zonas circunvecinas a éllas. El riesgo de enfermedad-maternidad cubre en 1967 a 230,762 asegurados y 409,882 familiares; se limita a los trabajadores que ganen un salario máximo anual de 14,400 Bs. (4.300 dólares). El riesgo de accidentes industriales y enfermedades profesionales cubre en 1957 y en las mismas zonas a 254,982 asegurados. Para este riesgo no hay límite del salario devengado, todos los trabajadores de la zona de aplicación están cubiertos.

El cuarto factor de importancia en lo relativo a salud ocupacional ha sido la labor del Ministerio de Sanidad y Asistencia Social. De modo directo, el Ministerio tuvo hasta 1954, como labor en salud ocupacional, los certificados de salud. De acuerdo con el reglamento para ciertos oficios se practicaba este certificado. En realidad es un triaje de tuberculosis y sifilis por sus dos pruebas básicas: la radioscopia y luego la fotofluoragrafía del tórax y la reacción de Kahn. Este examen se hacía cada año y en 1957 se otorgaron 255,464 de certificados. La población activa del país es de tres millones y medio de habitantes.

El año de 1954, es fecha de gran impotancia para la salud ocupacional en el Ministerio de Sanidad, pues élla corresponde a la creación de la primera organización específica del Despacho, con la colaboración del Instituto de Asuntos Inter-americanos, quien proporcionó el personal técnico comenzaron estas labores.

El personal de higienistas industriales (ingenieros) y de ingenieros químicos ha determinado la característica de la labor desarrollada. Un survey de industrias que alcanza en la actualidad a 455 con un total de 40,593 trabajadores representa el importante trabajo de esta sección.

Su ubicación administrativa está como Sección de Salud Ocupacional, en la División de Ingeniería Sanitaria, de la Dirección de Salud Pública. Se ha realizado un buen trabajo el cual ha sido objeto de publicaciones tales como la de Schmidt (3) y debemos considerar lo atinado en la selección del nombre para la sección. Nos parece el término de salud ocupacional, superior a los de medicina del trabajo, medicina industrial, medicina ocupacional, por calificar una actividad no solamente de modo más amplio, sino responder al derecho a la salud establecido en nuestra constitución.

Las posibilidades de desarrollo que son ya realidad con motivo de la reorganización del Ministerio de Sanidad son excelentes. Se ha creado un Departamento de Higiene del Adulto y en él las Divisiones de Salud Ocupacional y de enfermedades degenerativas, cáncer y enfermedades venéreas.

La labor normativa, de investigación y de preparación de personal que son las básicas asignadas a las Divisiones del Ministerio, tienen mayor amplitud en salud ocupacional pues tendrán que coordinar una serie de actividades actualmente diseminadas en varios ministerios e institutos autónomos. Como estas actividades dependen de la relación de estos organismos con empresas independientes, la labor es compleja. No puede limitarse a un aspecto burocrático, sino a utilizar lo más pronto posible los servicios médicos existentes a fin de planificar investigaciones de conjunto para el conocimiento del problema.

En el trabajo anteriormente citado mencionaba entre las conclusiones algunos que por ser ilustrados de esta exposición, creo conveniente repetir aquí. Además considero que algunas de éllas indican los elementos indispensables para el desarrollo del programa de salud ocupacional.

Las sumas perdidas por morbosidad y mortalidad de los trabajadores se estima en 200 millones de bolívares (60 millones de dólares) y es equivalente a una huelga de setenta mil trabajadores, hecho que debe tomarse en consideración para el desarrollo de los presupuestos.

La evolución por etapas de acuerdo con las poblaciones o las facilidades existentes deben regir el desarrollo del programa. Los sitios que poseen Unidades Sanitarias de primera clase y el desarrollo de investigaciones en los servicios médicos privados deben constituir las etapas iniciales y de especial importancia en este programa.

Entre los aspectos de investigación científica encomendados a la salud ocupacional la revisión de lo que actualmente se tiene en nuestras estadísticas y las pautas necesarias para darle valor a estos datos y poder utilizarlos en futuros análisis son de una absoluta necesidad.

No debería el Ministerio abocarse a establecer una legislación, sino haciendo uso de la amplia ley de sanidad vigente dictar mediante resoluciones ejecutivas medidas específicas para ciertas situaciones. El mecanismo de resoluciones es mucho más sencillo, permite la prueba experimental y su revisión periódica.

El quinto factor que debemos analizar es él de las empresas privadas. Su labor ha sido mínima en relación con su número de trabajadores. Hay una característica que no debemos pasar inadvertida como es la de incorporación de trabajadoras sociales. En algunas empresas no existen enfermeras, ni auxiliares de enfermería, pero existen médicos a tiempo parcial y trabajadoras sociales. Me parece interesante señalar esta característica que podría indicar el espíritu que anima al establecimiento de los servicios. El valor de la experiencia es de importancia, pues las trabajadoras sociales tratan de coordinar los servicios de la empresa con los del Seguro Social, obteniendo citas y manejando la tramitación burocrática, evitando un número de horas perdidas por el trabajador y logrando una mayor rapidez en la prestación misma del servicio.

Como sexto y último factor nos corresponde mencionar al gremio médico venezolano. Desde el punto de vista científico las publicaciones existentes en nuestra bibliografía nacional han sido aisladas y el movimiento de una organización científica de la salud ocupacional comienza con la creación de las Sociedades de Medicina del Trabajo y del Deporte y Salud Pública.

La Sociedad Venezolana de Medicina del Trabajo y del Deporte se instala en 1950 y sus miembros son autores del mayor número de publicaciones sobre el tema. Si la labor ha sido dificil y no ha tenido el rendimiento esperado, sin embargo no podemos de dejar de mencionar el éxito alcanzado en la realización del Tercer Congreso Americano de Medicina del Trabajo, reunido en Caracas en noviembre de 1955.

La Sociedad Venezolana de Salud Pública, fundada en 1952, no tiene todavía sección especializada en Salud Ocupacional y en sus asambleas se han presentado trabajador relativas a esta especialidad.

El campo de organización científica de profesionales relacionados con la salud ocupacional es más que suficiente en ambas sociedades y es de esperarse un desarrollo progresivo de actividades.

El aspecto gremial de los médicos en relación con salud ocupacional es muy favorable. Ya hemos mencionado que no existe una oposición sistemática. Hace dos meses se firmó un contrato de trabajo entre la Federación Médica Venezolana y el Instituto Venezolano de los Seguros Sociales, basado en el hecho de no ser considerados empleados públicos. Este hecho, no ser empleados públicos, dificulta cualquier centralización por parte del Gobierno de sus propios servicios, no solo relacionados con salud ocupacional sino también con servicios médicos al pasar al Ministerio de Sanidad en condición de empleados públicos perderían su libertad de contratación de trabajo y quedaron sometidos únicamente a los azares políticos.

La labor actual de la Federación Médica Venezolana es sumamente dificil. Los organismos médicos gremiales no están organizados para responder ,a las múltiples necesidades que se han ido acumulando y pueden encontrarse frente a realidades que por esta falta organizativa no habían previsto y les será más dificil corregir. La salud ocupacional toca con los problemas más variados e internos del ejercicio profesional y requiere una actitud vigilante y rápida acción. No podrán existir programas de salud ocupacional que se desarrollen armoniosamente si en éllos se ha prescindido del médico, uno de los profesionales más importantes para la buena administración de los servicios y el logro de los objetivos.

Hemos presentado seis factores relacionados con la salud ocupacional: legislación laboral, empresas petroleras, Seguro Social, Ministerio de Sanidad, empresas privadas y médicos, que a nuestro juicio y en ese orden de importancia han jugado papel importante en su desarrollo.

Las posibilidades futuras están bajo los mismos grupos en época democrática el papel orientador del Gobierno adquire una especial importancia. Es necesario aclarar que la función orientadora del Gobierno no implica que creamos o recomendamos como conclusión inmediata la nacionalización de todos los servicios de salud ocupacional, como en cualquier otra actividad de patronos y trabajadores es más lo que puede lograrse mediante discusión de contratos colectivos y adecuada orientación de Gobierno que mediante una nacionalización que posiblemente tenga más de socialización demagógica que de programa de salud pública.

La filosofía de gobierno basada en la contribución de los ciudadanos y por ende de las empresas de acuerdo con sus posibilidades económicas permite la coexistencia de servicios privados y gubernamentales.

En los planes inmediatos, como es la prestación de ciertos servicios de salud ocupacional como medicina ocupacional y saneamiento del ambiente del trabajo la competencia de la empresa y sus trabajadores es preponderante. El Gobierno debe limitarse a imponer normas mínimas de prestación de servicios, goce de beneficios económicos y mantenimiento saludable del ambiente de trabajo.

En los planes mediatos o a largo plazo el Gobierno complementa lo existente mediante la provisión de seguros y prestaciones económicas y otras dentro del regimen de seguridad social, pero su misión en ningún caso es de sustituir, complementar cuando lo creyere conveniente a la total economía de la nación y crear.

El desarrollo de estos postulados requiere básicamente la existencia de regímenes democráticos, pues son éllos los únicos que permiten la planificación de actividades en beneficio de los ciudadanos.

Para las empresas estos mismos regímenes democráticos son los únicos convenientes a largo plazo. Una y mil veces han quedado demostrado que las ventajas económicas obtenidas durante la dictadura no tienen carácter permanente, dan origen a represalias de toda índole y siembran profundos desacuerdos que perturban las relaciones humanas y no desaparecen con la rapidez necesaria.

Hemos tratado de analizar los factores principales que han determinado la labor realizada en salud ocupacional y como podrían orientarse para el desarrollo de un programa adecuado a Venezuela. El análisis de estos factores tiene a mi juicio un valor modesto pero es indiscutible que el presentarlos en forma ordenada pueden servir de base a una discusión que no debe ser aplazada.

RESUMEN

El autor analiza a partir del comienzo del presente siglo los factores que a su juicio han jugado papel preponderante en salud ocupacional: legislación laboral, empresas petroleras, seguro social, Ministerio de Sanidad y Asistencia Social, otras empresas privadas y gremio médico. Considera el impacto que han tenido sobre estos factores los últimos diez años de dictadura que impidieron el armonioso desarrollo de actividades. La legislación laboral sirvió para establecer la prestación en accidentes y enfermedades profesionales y de permisos materiales, pero sobretodo para constituir bases mínimas para discusión de contratos colectivos. Se observa la labor desarrollada por el Ministerio del Trabajo y la necesidad de restringirla al control de la aplicación de la legislación. El papel de las empresas petroleras se presenta en sus aspectos negativos y positivos. El tratar de imponer fórmulas de otros países en los programas de salud ocupacional venezolana es ignorar la realidad. El desarrollo del Seguro Social a partir de 1944 ha traido beneficios e interferido en forma desfavorable al desarrollo de los servicios médicos de las empresas petroleras.

La coordinación de sus labores y los cambios de su legislación son indispensables. Se presenta la nueva orientación del Ministerio de Sanidad y Asistencia Social, quien debe asumir la mayor responsabilidad en estos programas. La labor de otras empresas privadas ha sido muy reducida y como factor característico tiene él de la incorporación de las trabajadoras sociales. Se estudia la actitud del gremio médico venezolano y se considera que es favorable al desarrollo de este tipo de programas siempre que se mantengan principios calificables como mínimos: contratación, respeto de la relación médico-paciente, alto nivel de personal e instalaciones. Se concluye la necesidad de establecer programas en el Ministerio de Sanidad y Asistencia Social y se reafirman los beneficios que para las empresas y trabajadores tiene la existencia de régimenes democráticos, los cuales permiten la coexistencia y armonioso desarrollo de los servicios privados y gubernamentales en beneficio de la salud ocupacional.

SUMMARY

The author analyses the factors that in his opinion have played a predominating role in the evolution of occupational health in Venezuela since the beninging of the present century: labor legislation, the oil companies, social security, the Ministry of Public Health, other private enterprises and the Medical Association. He examines the havoc wrought on these factors by the last ten years of dictatorial regime which interfered with the harmonious progress of activities. Labor legislation brought about the establishment of services and benefits in cases of industrial accidents and illnesses and leaves of absence in cases of maternity; its most important significance was, however, the use of its provisions as minimum bases for the negotiation of collective contracts. Attention is given to the work performed by the Ministry of Labor and to the advisability of restricting it to controlling the application of the labor legislation. The role of the oil companies is set forth in its positive and its negative aspects. To try to impose systems from other countries is to ignore reality. The development of social security since 1944 has had many beneficial results, but it has also interfered unfavorably with the development of the medical services of the oil companies. Coordination of activities and change in legislation are indispensable measures. The author comments on the new trend of policies of the Ministry of Public Health, which should assume a larger responsibility in these programs. The work of other private enterprises has been very limited and its characteristic feature has been the introduction of social workers. A study of the attitude of the Venezuelan Medical Association leads to the conclusion that it is favorable to this type of programs, provided that principles which may be considered as minimum regiments are maintained: negotiation of contracts, respect of the doctor-patient relation and a high level for personnel and facilities. The general conclusions arrived at are the need for establishing programs in the Ministry of Public Health and the favorable influence, both for companies and workers, of the existence of a democratic regime which permits the coexistence and harmonious development of private and governmental services to the advantage of occupational health.

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CAMPANHAS À BASE DE INSECTICIDAS CONTRA INSECTOS TRANSMISSORES DE ENFERMIDADES TROPICAIS COM EXCEPÇÃO DA MALÁRIA

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1. — INTRODUÇÃO

Coube-me a tarefa de relatar sobre as campanhas à base de insecticidas contra as espécies vectoras de doenças tropicais.

As limitações de tempo aqui estabelecidas para uma apreciação das inter-relações entre esses insectos, os homens e os agentes etiológicos é um campo tão vasto que nos perdemos já antes de penetrá-lo.

Com referência ao homem, sua actividade constante é destinada a suprir as necessidades essenciais para sua subsistência, como os demais seres; porém, além desse objectivo, ele tem a capacidade de construir e proteger-se contra os extremos da acção dos agentes físicos e climatológicos da natureza; essa protecção poderá ser individual, com a cobertura directa do próprio corpo, ou se amplia e realiza uma separação de espaço, construindo para si um invólucro protector e correctivo, a fim de controlar as variações físico-climatológicas externas, que na sua origem são incontroláveis.

A protecção contra os efeitos do sol e da chuva tem sido a constante preocupação do homem desde a sua primitiva cabana.

O microclima do ambiente formado pelo abrigo, ao condicionar os excessos de variações da natureza, cria condições mais favoráveis à vida do homem, e, por outro lado, essa relativa estabilidade climática cria também ambiente propício à vida de outros seres, que assim passam a viver com ele em sociedade.

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2. — O HOMEM E A SUA ECONOMIA

A sociedade estabelecida com o homem, promovida pela aproximação desses seres vivos, pode — e com frequência — criar uma integração, estabelecendo um novo estado de vivência.

Ora, como todo animal, o organismo humano funciona em razão das trocas energéticas, como qualquer máquina, queimando o carbono e realizando um trabalho que poderia ser medido pela diferença entre o seu calor interno e externo.

Porém, com a associação de seres irracionais, grande parte da energia utilizada pelo homem é desviada para o metabolismo desses agentes e a actividade resultante fica reduzida, chegando a ser eliminada, indo até a eliminação total da actividade do homem.

A verdade dessas afirmações é confirmada pelos dados estatísticos oferecidos pela tabela I, onde podemos constatar que para os países de uma maior vida média de seus habitantes corresponde um maior desenvolvimento económico, caracterizado pela maior renda *per capita* de suas populações.

Os dados estatísticos sobre a mortalidade são mais fàcilmente registrados; porém, não devemos esquecer que os casos mórbidos, embora de difícil mensuração, é evidente que provocam anemias de graus variáveis, desnutrição, caquexia, etc., e o homem com essas enfermidades, e como elemento essencial no mecanismo da produção, não pode exercer a plenitude de sua actividade nesse estado deficitário de energia. Essa redução de capacidade de trabalho reflecte-se consequentemente no poder económico da região.

3. — OS INSECTICIDAS

A transmissão da maioria das doenças que têm os insectos como vectores é uma consequência da necessidade que eles têm de alimentar-se de sangue, indo procurar esse alimento — com mais vantagens ao abrigo das intempéries — onde geralmente o homem também repousa e permanece longo tempo.

Para interromper a transmissão, uma das soluções evidentes é eliminar os insectos do convívio do homem: essa tarefa hoje está

QUADRO I

Países	Renda *per capita* (U. S.)	Estimativa de vida ao nascer	Taxas brutas de mortalidade por 100 000 habitantes (1956)	Taxa de mortalidade segundo as causas, por doenças transmissíveis, para 100 000 habitantes			
				Peste	Tifo-*	Malária	Outras-**
Estados Unidos	1 870	67 (1955)	930 (1955)	—	—	—	2,6
Canadá	1 310	66 (1952)	821 (1956)	—	—	—	2,4
Nova Zelândia	1 000	68 (1952)	896 (1955)	—	—	—	4,2
Bélgica	800	62 (1949)	1 226 (1955)	—	—	—	4,0
Inglaterra	780	67 (1955)	1 167 (1956)	—	—	—	2,3
Dinamarca	750	67 (1950)	886 (1956)	—	—	—	3,2
Noruega	740	69 (1950)	849 (1955)	—	—	0,1	2,9
Alemanha (Oeste)	510	65 (1953)	1 099 (1955)	—	—	—	2,7
Holanda	500	71 (1955)	747 (1954)	—	—	—	2,8
Argentina	460	56 (1947)	819 (1954)	—	0,1	0,2	7,0
Chile	360	49 (1952)	1 281 (1954)	—	1,0	—	10,3
Itália	310	53 (1932)	929 (1955)	—	—	0,1	5,8
União Sul-Africana	300	41 (cor)	1 762 (1954)	0,2	0,1	—	16,0
Colômbia	250		1 329 (1956)	—	7,0	12,7	44,9
Brasil (DF)	230	49 (1951)	1 205 (1954)	—	—	0,3	33,7
Japão	190	63 (1955)	804	—	—	—	7,0
Equador (Quito)	150	50 (1951)	1 550 (1955)	2,3	0,6	38,2	44,6
Paraguai	140		1 060 (1950)	—	—	—	—
Peru (Lima)	120	46 (1943)	404	—	0,1	1,7	7,1
Egipto	120		1 840 (1953)	—	0,1	0,1	9,2
Paquistão	70		1 190 (1951)	—	—	—	—
Índia	60	32 (1941)	2 740 (1950)	—	—	—	—

OBSERVAÇÕES:

Tifo-* = Tifo e outras doenças por ricketzia.

Outras-** = Todas as doenças transmissíveis de classificação pouco precisa, entre as quais estão as demais doenças transmissíveis por artrópodos.

Fonte — Fundo Monetário Internacional e Demographic Yearbook, 1957, United Nations.

bastante facilitada, com o advento dos insecticidas de acção tóxica residual, pelo poder letal que possuem sobre uma grande variedade de insectos, protegendo o homem, sem prejudicá-lo, por lhe ser inócuo nas dosagens utilizadas.

A técnica da borrifação com insecticidas tem sido generalizada, principalmente em programas de *contrôle* de malária, porém sua eficiência se verifica para quase todas as doenças transmitidas por artrópodos, principalmente quando a transmissão é intradomiciliária; graças a essa técnica, a malária reduziu a índices inexpressivos nas estatísticas de saúde, onde sempre ocupou posição destacada.

Devido à importância da malária e à grande dispersão de sua incidência nas várias partes do mundo, o D.D.T. tem sido utilizado em grande escala, cobrindo uma extensa área, graças ao *contrôle* eficiente dos anofelinos, realizado com esse insecticida. Acontece, porém, que os domicílios onde se abrigam os anofelinos abrigam também outros insectos sensíveis à acção do D.D.T. e por isso as campanhas realizadas contra a malária têm igualmente contribuído para o combate aos outros insectos transmissores e, por conseguinte, as outras doenças têm sido beneficiadas com esse processo. Por falta de qualquer dado estatístico ou outros elementos informativos, não podemos medir a eficiência da técnica de borrifação intradomiciliária com relação a esses outros insectos. São esses os efeitos colaterais obtidos com as campanhas de malária assinalados por Gabaldon.

Resistência aos insecticidas. — A evolução do processo da imunidade domiciliária pelo tratamento químico tem trazido suas apreensões. O tratamento continuado das casas, com insecticidas, tem revelado que alguns insectos ficaram insensíveis aos efeitos tóxicos, tão evidentes nos primeiros tratamentos. Nos primeiros anos de uso dos insecticidas residuais, em 1946, tornou-se conhecida uma manifestação de forma de resistência aos insecticidas clorados e em 1955 já eram conhecidas 37. A mosca foi uma das primeiras a revelar essa resistência; porém, com a intercalação com outros insecticidas e adição de substâncias sinergéticas, vão surgindo novas esperanças e o resultado é que as condições sanitárias continuam melhorando, talvez não tão râpidamente como desejávamos.

4. — *CULEX FATIGANS* — FILÁRIA

Extensão da doença. — Stoll estima que, provàvelmente, devemos ter trezentos milhões de doentes de filária em toda região tropical, se estendendo desde 40° de latitude norte a 30° de latitude sul, em todos os continentes, menos na Europa.

No Brasil já foram registados casos de filária autóctone em 7 cidades, capitais de Estado, sendo Belém do Pará a cidade onde foi assinalada a maior incidência dessa helmintose.

Os transmissores e seus hábitos. — A maior responsabilidade pela transmissão da filariose (bancroftose) no Brasil tem sido atribuída ao *Culex pipiens fatigans*, embora tenham sido encontrados infectados o *A. darlingi* e o *A. tarsimaculatus*.

Os hábitos domiciliários do *Culex fatigans* possibilitariam um *contrôle* eficiente da doença, sòmente com a aplicação de insecticida no interior do domicílio, mesmo porque logo após a primeira aplicação, há um desaparecimento desses mosquitos do interior das casas; entretanto, logo em seguida, eles se tornam insensíveis à acção do insecticida.

Fazendo-se uso do *Dieldrin*, não foi verificado qualquer resultado favorável, e os hábitos de pouso do mosquito foram alterados, porque, enquanto 80 % dos mosquitos pousavam nas paredes antes do uso do *Dieldrin* e sòmente 20 % nos móveis, depois da borrifação com esse insecticida 20 % passaram a pousar nas paredes e 80 % nos móveis.

A *W. malayi* é uma doença rural porque o seu transmissor, do grupo mansonóide, se encontra de preferência nessas áreas.

O processo mais eficiente para o *contrôle* do *Culex* tem sido pelo combate intensivo na fase larvar.

5. — FLEBÓTOMOS (LEISHMANIOSE)

Hábitos do transmissor. — Dentre os transmissores, as espécies que melhor índice de transmissão têm apresentado são, naturalmente, aquelas cujos hábitos domiciliários são mais pronunciados, apesar de serem os flebótomos de características silvestres bem acentuados. As casas onde foram encontrados os flebótomos são em geral de barro e o piso de terra, na zona rural, tendo quase sempre animais domésticos em torno da casa, e seus habitantes apresentam nível de vida baixo, portanto situação muito primitiva de vida. Durante os períodos de chuva o número de flebótomos dentro de casa (abrigo) é tão abundante que necessitam afugentá-los para poderem dormir.

As fêmeas do *P. longipalpis*, embora de hábitos alimentários eclécticos, apresentam um antropofilismo bastante acentuado, sugando o homem a qualquer hora do dia, principalmente dentro de casa, mas podendo sugá-lo também até fora do domicílio. Deane afirma que tudo indica que o flebótomo entra na casa para sugar e não meramente à procura de abrigo. O facto é que as capturas domiciliárias são, geralmente, abundantes nas áreas dos flebótomos. Na Índia o transmissor responsável é o *P. longipalpis* e na China o *P. chinensis*. No Mediterrâneo o transmissor responsável é *P. perniciosus;* prefere alimentar-se de sangue bovino e canino, sendo o homem preterido na presença desses dois animais.

Combate ao transmissor. — No caso do *P. longipalpis*, no Brasil com hábitos domésticos bem pronunciados, a aspersão do insecticida D.D.T., na dosagem de 2 até 15 g/m^2, foi o suficiente para fazê-lo desaparecer completamente do interior das habitações durante 4 meses de observação, enquanto que nas casas não tratadas a densidade intradomiciliária do *P. longipalpis* continuou normalmente.

Em face dos resultados obtidos nas provas experimentais, foi realizado um esforço para ampliar a desinsectização da área malarígena já realizada rotineiramente e estendê-la a outras áreas afectadas sòmente pela leishmania; assim é que:

Em 1954	foram	desinsectizadas	27 707	casas
Em 1955	»	»	15 942	»
Em 1956	»	»	32 640	»
Em 1957	»	»	63 580	»

6. — TRIATOMÍDEOS — DOENÇA-DE-CHAGAS (TRIPANOSSOMÍASE AMERICANA)

Extensão da doença. — Têm sido diagnosticados casos em todos os países das Américas, com maior ou menor gravidade, menos no Canadá.

Hábitos e capacidades dos transmissores. — A característica principal dos triatomídeos, e que faz com que sejam os transmissores, é o hábito alimentar, em primeiro lugar, por ser um hematófago obrigatório e muito voraz; em segundo lugar, porque algumas das espécies têm hábitos exclusivamente domiciliários, como acontece com o *Triatoma infestans*, no Brasil.

As espécies silvestres são de menor importância na transmissão da doença para o homem, uma vez que poderão alimentar-se de sangue e outros animais, sem necessitar procurar o homem como sua fonte de alimentação.

Dos inquéritos realizados no Brasil de 1950 a 1955, para o conhecimento da doença, foram encontrados os seguintes resultados, por índices de infecção e por espécies de triatomídeos:

Triatomas infestans (75 661 exemplares examinados)	27,69 %
Panstrougilus megistus (25 173 exemplares examinados). . . .	11,28 %
Triatoma sordida (5 907 exemplares examinados).	9,65 %
Triatoma brasiliensis (1 396 exemplares examinados)	8,52 %
Triatoma maculata (949 exemplares examinados)	3,27 %

Combate ao transmissor. — Sendo uma doença sem qualquer possibilidade ainda de recursos terapêuticos específicos, por conseguinte de cura dos casos humanos, o único meio de reduzir o número de doentes é sem dúvida impedir a transmissão.

Foi iniciada em 1950 a aplicação extensiva dos insecticidas ao combate dos triatomídeos. Numa área do Estado de Minas Gerais empregou-se B.H.C. e *Rodiathox* (fosforado), compreendendo 125 municípios, com uma superfície total de 154 144 km^2 e uma população de 1 941 953 habitantes (censo de 1950). No Estado de São Paulo a área incluída compreendia 104 municípios, com uma superfície total de 74 491 km^2. Dessa experiência ficou demonstrado que os dois insecticidas se equivalem tècnicamente, ficando a escolha de um ou de outro fundamentada em razões económicas ou práticas. O *Rodiathox* apresenta, além do custo, o inconveniente de sua elevada toxicidade ao homem, o que obriga a certas precauções da parte do pessoal operador.

Em 1954 foi também iniciada uma campanha em base experimental, para estudo comparativo da eficiência entre o B.H.C. e o

Dieldrin, à semelhança do que foi realizado com o tiofosfato (*Rodiathox*). O B.H.C. utilizado foi ainda o pó molhável com 12 % de isómero gama e o *Dieldrin* também sob a forma de pó molhável com 50 % de princípio activo, dando às casas tratadas uma concentração de 0,5 g e 1,0 g por metro quadrado, de princípio activo respectivamente para o B.H.C. e o *Dieldrin*.

No segundo semestre de 1954 e primeiro semestre de 1955, foram tratadas 114 400 casas com *Dieldrin* (no Estado de Goiás). Quanto aos resultados obtidos pelos dois insecticidas contra o *T. infestans*, apresenta-se o seguinte quadro, organizado por Bustamante *et al.*:

Quadro II — *Efeito do* Dieldrin *(1 g/m2) e do* B.H.C. *(500 mg de isómero gama por metro quadrado) sobre o* T. infestans.
Data da aplicação dos insecticidas — Abril de 1954

Datas das inspecções (mês e ano)	Área tratada com *Dieldrin*		Área tratada com B. H. C.	
	Percentagem de casas infestadas	Número médio de triatomideos por casa	Percentagem de casas infestadas	Número médio de triatomideos por casa
Março de 1954 (1 mês antes da aplicação dos insecticidas	78,3	17,6	91,8	23,0
Julho de 1954	47,7	7,6	6,8	0,3
Novembro de 1954	64,1	29,1	31,1	2,0
Fevereiro de 1955.	78,4	13,6	25,6	1,5
Abril de 1955	75,0	18,6	27,5	2,3

De onde concluímos ser o B.H.C. mais eficiente contra o *T. infestans*, sem apresentar qualquer inconveniente de toxicidade ao homem, nas dosagens utilizadas.

Carrillo (1954) na Venezuela, Peñavel e Villagran (1956) na Guatemala, fazendo uso do *Dieldrin* contra outra espécie — o *Rhodnius prolixus*, mesmo o *T. dimidiata* (Guatemala) — obtiveram uma redução no índice de infestação domiciliária de 100 a 10,2 % após 7 meses, e nas áreas sòmente com *R. prolixus* uma redução de mais de 95 % após 2-3 anos.

Bustamante e Barreto, em experiências realizadas em Itaverá, em um total de 679 casas onde foi aplicado o B.H.C. (pó molhável) na concentração de 720 a 1 400 mg/m², isómero gama, chegaram às seguintes conclusões: 1.º) que três aplicações de B.H.C. em casas infestadas, com intervalo de 4 meses entre as aplicações, pode erradicar o *T. infestans*; 2.º) Que uma única aplicação conveniente de B.H.C. em casas infestadas reduz enormemente o *T. infestans*.

7. — PULGAS — PESTE

Extensão da doença. — Todo o sul da Ásia e norte da China, norte da África, África Oriental, África do Sul, sul dos Estados Unidos e América do Sul.

Combate ao transmissor. — Das provas feitas para solucionar os insecticidas mais eficientes, foram tiradas as seguintes conclusões:

a) O cianogás tem um efeito reduzido no índice culicidiano;
b) O D.D.T. e o B.H.C. têm uma acção residual sensível até 84-90 dias sobre o índice culicidiano;
c) A acção residual do D.D.T. é maior que a do B.H.C.;
d) A redução do índice pelo B.H.C. é mais sensível.

De 1954 a esta parte, introduziu-se na luta contra a peste o B.H.C., cujas propriedades pareciam mais vantajosas, especialmente por sua acção mais enérgica, daí porque passou-se então a empregá-lo de mistura ao D.D.T., na proporção de 0,5 g/m², e em suspensão na água. Tal factor trouxe a grande vantagem de fixá-lo melhor ao sol, sendo mais difícil a sua remoção rápida pelo pisoteio. Na aplicação procurava-se empregar a dose de 3,0 g/m² de piso.

Por este processo pode-se interromper o ciclo de transmissão da peste, e, em todas as ocorrências havidas, os casos foram em número bastante reduzido.

Despulização das casas — Em 1956 foram despulizadas 551 211 casas e 517 431 em 1957.

8. — *AEDES AEGYPTI* — FEBRE-AMARELA

As companhas contra o *Aedes aegypti* visaram de início principalmente a fase larvar do mosquito. O D.D.T. usado como larvicida pode ser usado com maior facilidade em extensão, uma vez que as doses empregadas são inteiramente inócuas para o homem e bastante tóxicas para as larvas; ao mesmo tempo que se faz o uso como larvicida, faz-se também como adulticida. Esse método tem sido bastante eficiente, não sòmente para controlar, porém mesmo chegando a erradicar o mosquito, como aconteceu no Brasil, embora a extensão territorial represente a metade do continente sul-americano.

A Bolívia também está sem *Aedes aegypti* desde 1948. A Guiana Francesa desde 1952. O Chile, Peru, Equador, Paraguai, Uruguai e Guiana Britânica estão com os trabalhos bem adiantados para a erradicação.

9. — ESCORPIÃO — ESCORPIONISMO

Embora tratando-se de um aracnídeo, convinha aqui relatar sucintamente a campanha realizada em Belo Horizonte, capital do Estado de Minas Gerais, no Brasil, onde em 1951 foram registadas mais de 100 pessoas picadas e 13 casos de óbitos até Outubro.

Hábitos de capacidade do artrópodo. — A caracterização do problema ficou melhor elucidada quando se estudou melhor o aracnídeo, cuja espécie é o *Tityus serrulatus*, de hábitos domiciliários bem pronunciados e possuidor de um veneno com uma potencialidade letal bastante maior que as outras espécies.

Combate ao artrópodo. — Verificando-se a sensibilidade desses aracnídeos aos insecticidas, ficou constatado ser o B.H.C. de grande eficiência na dosagem de 300 mg de isómero gama por metro quadrado, tendo uma acção letal quase instantânea.

Na primeira campanha realizada em Belo Horizonte foram borrifadas 78 146 casas e 8 933 quintais, conseguindo-se com essa providência a ausência total de óbitos e raras e imprecisas picadas; nos anos seguintes foram realizadas sòmente pesquisas e eliminação de focos isolados.

Campanhas realizadas no Brasil — Sumário de algumas endemias transmitidas por artrópodos

Especificação	1956	1957
Doença-de-chagas:		
Unidades da Federação trabalhadas	8	8
Municípios trabalhados	229	255
Levantamentos:		
Municípios trabalhados	17	103
Localidades trabalhadas	4 934	4 399
Prédios trabalhados	41 216	87 639
com triatomídeos	4 550	4 483
Triatomídeos capturados	31 282	25 802
Pó insectífugo consumido (quilos)	686	976
Expurgos:		
Municípios trabalhados	—	169
Localidades trabalhadas	16 606	7 673
Prédios expurgados	286 857	219 495
Consumo de insecticidas (quilos)	228 430	197 292
Febre-amarela:		
Unidades da Federação trabalhadas	25	25
Combate ao Aedes (Stegomya) aegypti:		
Municípios trabalhados	162	79
Localidades trabalhadas	10 759	1 254
Inspecções prédios	4 774 701	2 284 300
depósitos	34 271 668	21 720 419
Prédios com focos de *Aedes* (*Stegomya*) *aegypti*	—	—
Filariose:		
Unidades da Federação trabalhadas	6	12
Profilaxia:		
Municípios trabalhados	3	26
Localidades trabalhadas	3	42
Prédios trabalhados	49 971	7 911
desinsectizados	35 492	—
Depósitos trabalhados	1 208 209	934 160
desinsectizados	93 938	339 604
Galerias pluviais inspeccionadas (metros)	—	—
Valas inspeccionadas (metros)	78 226	3 856

Especificação		1956	1957
Insecticida gasto	Óleo (litros)	252 544	150 226
	D. D. T. (quilos)	11 389	15 453
	B. H. C. (quilos)	—	6 159
Leishmaniose:			
Localidades trabalhadas		3	3 009
Unidades da Federação trabalhadas		3	5
Municípios trabalhados		68	115
Casos		310	299
Cães examinados	número	7 278	32 731
	positivos	102	263
Flebótomos capturados		13 067	24 223
Medicações		20 036	29 028
Exames serológicos		1 710	1 497
Prédios dedetizados		32 640	63 580
Peste:			
Unidades da Federação trabalhadas		10	8
Municípios trabalhados		167	105
Localidades trabalhadas		—	5 996
Inspecções de prédios		2 288 875	1 097 755
Despulizações		1 950 885	783 099

Em relação ao ano de 1957, os dados são estimados.

10. — MOSCAS — DOENÇAS DIVERSAS

Embora os pesquisadores das ciências naturais tenham provado que a transmissão de certas doenças se faz por intermédio de alguns artrópodos, já em 1587 um arguto historiador português, de nome Gabriel Soares de Sousa, observou no Brasil que a mosca era transmissora de doenças e no *Tratado Descritivo do Brasil* assim ele a descreve: «Digamos logo, dos mosquitos, a que se chamam de nitinga, que são muito pequenos e da feição das moscas, os quais não mordem, mas são muito enfadonhos porque se põem nos olhos e nos narizes e não deixam dormir de dia, no campo, se não faz vento. Estes são amigos de chagas, e chupam-lhe a peçonha que têm e se vão pôr em qualquer coçadura de pessoa sã, deixam-lhe a peçonha nela do que se vêem muitas pessoas a se encher de boubas.»

McGuire capturou moscas nas faces das crianças no Egipto, encontrando nessas capturas uma predominância da *Musca doméstica ricina* e *Musca sordens;* e, do material capturado, foi isolada e estudada a flora bacteriana da superfície externa dessas moscas, assim como as bactérias do seu interior. Em ambas as espécies foi encontrado no seu interior um número de bactérias 20 vezes maior (aproximadamente) de que no seu exterior. Dentre esses organismos foram encontrados, como dos mais numerosos: *Coli-aerogens, Estreptococus, Staphylococcus, Bacilos, Clorstridium, Corynobacterium, Hemophilus, Neiseria, Sarciva.*

Cox (1912) e Scott (1917) já se referiam ao grande número de bactérias transportadas pelas moscas, principalmente nas cidades sem condições sanitárias. Flogel (1953) verificou ainda no Egipto que 11 % das moscas transportavam *Salmonella* ou *Shigella,* agentes patogénicos das doenças entéricas do homem.

Têm sido igualmente encontrados, na flora bacteriana transportada pelas moscas, agentes etiológicos de várias doenças respiratórias e infecções piogénicas e, em alguns casos, o *N. gonorrhoeae* e o bacilo de Koch-Weeks.

McGuire observou, ainda, que os organismos do grupo *micrococcus* são isolados com frequência no interior das moscas, o que faz supor ser a mosca um agente vector não sòmente mecânico, mas também de natureza biológica, reproduzindo bactérias no seu interior.

Combate ao transmissor. — As experiências satisfatórias obtidas com a borrifação da mistura de vários insectidas, principalmente *Diazinon,* mesmo em baixas concentrações de tóxicos, como foi observado por Gahan, na concentração de 0,1 %, a mortalidade de moscas foi de 99 %, algumas vezes de 90 %, aumentando a mortalidade com maior concentração de insecticida e permanecendo seu efeito por duas ou três semanas. Essas provas foram feitas em ambiente onde havia também moscas resistentes.

Em face da preferência das moscas pelo pouso em superfícies lineares, como seja fios de cordas pendurados, empregaram cordéis com *Diazinon,* conseguindo-se um *contrôle* durante 6 a 8 semanas, e, quando retiraram o *Diazinon,* as moscas invadiram o ambiente, crescendo de 6 para 133 moscas. Reinstalado o *Diazinon*, o índice baixou para 4. Campanhas semelhantes têm sido realizadas com o *Malathion*.

Resultados obtidos. — Corbo, na Itália, constatou que, em áreas onde se fazia o tratamento rotineiro das casas com D.D.T e outros insecticidas clorados, em mais de 8 000 casas, para o combate à malária, a redução da mortalidade infantil foi bastante pronunciada.

Gabaldon, na Venezuela, observou, em áreas tratadas com o D.D.T intradomiciliário, que o índice de mortalidade baixou de 6,7 pontos, enquanto em outras áreas de igual nível a redução foi sòmente de 3,3 pontos.

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SOME EXPERIENCES OF W.H.O. IN ENVIRONMENTAL SANITATION

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As an engineer in such a sea of medicos I should be lonely. As a practical sanitation salesman in this distinguished company of scientists I should feel an overwhelming inferiority. I have neither sensation, for the conditioning of a lifetime of work alongside the people who compose public health teams makes me feel quite at home in such company. I want to discuss with you for a few minutes a subject which is completely narrative and non-scientific — some experiences of the World Health Organization in its environmental sanitation activities and some of the needs and challenges which lie ahead.

HOW W.H.O. OPERATES

Contrary to a great body of public opinion, the World Health Organization is not a supra-national health department. It does not exercise any police power. It sets no mandatory standards or requirements; it does not operate by itself any health programme. You cannot call WHO over the telephone to have a dead dog removed from the street. Theoretically, it serves, only upon request, the needs and desires of its 88 Member States, but actually its secretariat exercises a considerable amount of initiative. It appraises the health needs of the world, it stimulates and guides countries to follow beneficial courses in their health development, and it assembles and

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makes available to them the best and latest scientific information in this field. Since it operates on a very small budget (less, for example, than it costs any big city to collect its garbage), WHO is not a «doing» type of organization. It does not build structures. It provides very few material things. It does not by itself carry on programmes of work. Instead, it endeavours to furnish information, inspiration, guidance and technical service to is member states, and to co-operate with them in their health projects and programmes.

Among the services which WHO attempts to provide and the fields of endeavour which it tries to stimulate, the control of the environment in behalf of health looms large in importance. Historically the environmental diseases have been man's greatest killers. While sanitary measures have reduced the mammoth proportions of the old scourges of cholera, plague, typhus, typhoid and yellow fever, most of them still persist in areas and pockets which are not blessed with means of protection. For example, there have been reported for 1958, up to the end of July, 48 729 cases and 20 687 deaths from cholera in five countries of South-East Asia. In the countries of the West where sanitation has brought such high reward in freedom from the enteric diseases, such comfort and convenience of living, it is likely to be overlooked that three-quarters of the people of this earth live a miserable existence in a quagmire of insanitation, suffering and dying from diseases spread by infected water, by insanitary disposal of faecal matter, by uncontrolled insect and animal vectors of disease and by unprotected milk and food. We may easily forget that hundreds of millions of the world's peoples are ill-housed, live in rude shacks, under palm fronds, in caves or in hovels. It is hard to believe that in one Eastern country 4000 people die every day as a direct result of water-borne diseases. It is ironical that in this same country one-quarter of the working hours of the housewives is spent in finding and bringing this foul drinking water. The cumulative effects of such labour, illness and death are factors of tremendous importance not only to the emotions of the people but to the country's economic welfare. It is to help alleviate such woeful conditions that WHO's first efforts are naturally directed.

A VARIED MENU

Although the consequences of insanitation in the unfortunate countries may be more spectacularly evident, with such countries having a first priority upon WHO's assistance, it must not be forgotten that great problems of sanitation exist in all countries, regardless of their stage of economic and social development. Sanitary problems may range in complexity from those at a Bedouin's temporary shelter to those of an atomic reactor. In fact, the problems of environmental control multiply mightily with the agglomeration of people and with the development of industry. Too often these growing problems go unrecognized in the pride that goes along with growth, and in the smugness that finds satisfaction in the safety of conditions that *used* to exist.

WHO must be in position to serve the needs of its entire clientele of nations, whether they be under-developed, developed or over-developed. Demands are being made upon it for a wide variety of services. From countries just starting the slow climb along the developmental curve the help requested is for the organization of sanitation services or the planning of programmes in the most elemental undertakings. Countries which have laid solid foundations in sanitation and have reached the steep section of the developmental curve, where much progress is being made in a short time, call for help on undertakings such as public water supply, sewage and waste disposal, food sanitation and the like. The far-advanced countries which have reached the top section of the ogive curve, where additional improvement comes slowly, may ask for aid in those complex problems accompanying urbanization and industrialization, such as water and air pollution and the safe disposal of radioactive wastes.

SOME THINGS LEARNED FROM EXPERIENCE

Environmental sanitation was singled out by the First World Health Assembly as one of the four most important health needs. Strong support has been given to this cause by every succeeding assembly and executive board, and the Director-General has taken advantage of every opportunity to stimulate and assist activity in this field. In spite of all these efforts, the *visible* effects of the accomplishment of WHO in this field during the first decade of its existence are not impressive. However, it is good to believe — to know — that the *invisible* and *intangible* values of this ten years' continuous effort are such as to yield satisfying returns in the future.

A great deal has been learned in these ten years about sanitation on the international scale. These new discoveries are probably not new at all, but derive from age-old traits of human conduct. At any rate they are exceedingly important to such work and other agencies working in the same field would doubtless report the same observations. Here are some of them:

In the first place, there is a general lack of understanding of the meaning of the word itself, and of the values of sanitation to health and life. In many quarters the term is considered to apply only to the disposal of human excrement — and that usually limited to the rural household. Among others sanitation is vaguely considered to be something that is good. an amenity rather than a necessity, perhaps a luxury for the wealthy or the effete — but in any case something which is beyond the economic reach of the impoverished man. It would be unfortunate enough if such ideas were limited to the unschooled; it is more serious to find them held by officials in responsible positions.

One might suppose that people would wish to do things which are good for them — especially after they find out such things *are* good for them. This is often not true. Instead, people continue to do the things that are habitual and traditional, or the things that satisfy the basic human insticts. They are interested in a water supply on the bases of convenience and appetite, not because it is safe. One of the most difficult public health tasks is to get people living in squalor to accept and to use decently a sanitary latrine.

From the beginning of WHO such interest as has been expressed in sanitation has been mostly in behalf of the vast rural populations of the world. It is understandable that delegates to the health assemblies should first interest themselves in these people. They are the people of the largest population group, many of whom live on the very margin of existence, and who are without leadership or resources to help themselves. By contrast, the urban dwellers are said to have a machinery of government, leadership and taxing power that may provide some facilities of sanitation.

In response to this demand, WHO's efforts in sanitation so far have been directed primarily toward rural sanitation. Most of the field projects have been of a type designed to stimulate rural sanitation or to demonstrate its practicability under prevailing conditions. Some of these projects have had good results and might be called worthwhile. Too many of them, alas, have not achieved the desired success. None of them has achieved that desideratum of public health officials: a spark to kindle the imagination and a spur to the emulative action of large numbers of people.

What are the reasons for falling short of objectives? First of all, there is poor justification any longer for «demonstration» projects. The potentialities of sanitation have long ago, and in thousands of places, been amply shown. What is needed is the secret of making practical and effective application of the facts already known. These projects, by their shortcomings, serve to say again and again the same truths that similar experiences should long ago have taught. These truths, to mention only four, are: (1) that rural sanitation undertakings isolated from, and unsupported by, well organized health departments do not start any chain reactions; (2) that there should be environmental sanitation *programmes* for a country, rather than *projects,* and that these programmes should have all the care of planning, design and execution as any other great undertaking; (3) that there must be qualified persons to perform such duties, they must be fired with a zeal to succeed, and they must be given the freedom of action and the support to make success possible; and (4) that improvement in rural sanitation is a very slow process, involving the understanding, acceptance and participation of the people, with a rate of change closely related to economic and social development.

RURAL VERSUS URBAN SANITATION

The sympathy of everybody goes out to the underprivileged and unorganized masses of rural people throughout the world, and the efforts to remedy their sad plight should not be relaxed. However, if emotion is discarded and sane epidemiological and sociological thinking substituted, the urgency in many situations will shift to the relief of *urban* insanitation. From the standpoints of disease tranmission and the degradation of human dignity, far greater peril lies in the crowded and grossly insanitary conditions in and around cities than in the dispersed villages and dwellings of the countryside. Communicable diseases may exist in families or settlements but they do not spread widely in the rural areas. By contrast, the opportunities for infection and dissemination of the environmental diseases are vastly greater in congested populations. Striking examples of this fact have been this year's epidemics of cholera in Calcutta and Bangkok.

In addition to epidemiological considerations are the practical potentialities of accomplishment. As we have seen from the discussion above, the rate of progress in rural sanitation is usually very slow and is geared to the advancement of educational, economic and social levels of the people. Moreover, there is lacking in such programmes any element of dramatic appeal to the people. No «benign contagions» spread over countries as the result of a popular urge to emulate the example set by demonstration projects. These desirable rural improvements come only through the careful planning and persevering attention of health departments. Environmental sanitation needs a dramatic product that will equal in human appeal those that are so effective in programmes of malaria eradication and yaws control.

There exists such an appeal in a programme of public water supply. The slogan might well be «Clean water for all the people». No activity could be closer to the life of the people. Nothing could bring greater comfort. Nothing could add more to the protection and promotion of health. Some other international agencies have experienced the magical acceptance of water supply and are profiting immensely from the contribution which safe and ample water supplies make to other health programmes, and to the economic development of the countries. In one country where an international agency promoted its first water supply project against great inertia 15 years ago, and where the project was paid for entirely by international funds, the news and popularity of this type of improvement have spread for and wide. In the meantime scores of systems have been completed and at present there are more than 400 designs for new water supplies upon the drafting boards. It is significant that the construction of these is being financed entirely from local and national funds.

As one travels about the world he finds similiar potentialities in many countries. Everywhere there is a demand for water — water that is clean and that does not have to be carried for endless miles on the heads of dutiful women. Surprisingly, there are almost everywhere—even in the most impoverished countries—considerable amounts of money available for water supply purposes. In many cases these funds are simply waiting for intelligent use, for officials do not know how to proceed and engineering talent does not exist for planning, design, construction and operational functions. In some cases, alas, the money is being wasted through predatory operators who take advantage of the innocence of public officials. Herein lies one of the greatest opportunities for WHO guidance and technical assistance. At a minimum of cost for advisory service and the orientation of key personnel, the Organization could become a catalytic agent of great usefulness. National governments and other interested international agencies might be expected to provide the great bulk of the money required for capital outlay. Working together, these agencies can launch a movement of great proportions and of widespread economic and social effect. This kind of benefit can be accomplished without the participation of the individual urban beneficiary.

History proves that there is no greater stimulus to human welfare than adequate water supply for domestic and industrial purposes. History as also demonstrated that most of the movements that have beneficied mankind have originated in populous urban areas and have diffused outward to affect the rural populations. Such may be the best course for sanitation. While maintaining a constant interest and pressure in behalf of the world's rural folk, it may be considerably more productive to redirect the present emphases of organizations such as WHO to the urban problems of environmental control.

PUBLIC HEALTH VERSUS PUBLIC WORKS RESPONSIBILITIES

Public officials are often confused by suggestions that public health agencies have anything to do with public facilities — even sanitary facilities — that involve structures. This is a field, they often say, which logically belongs to public works departments. This is quite true and is a principle that, in its usual and general aspects, is subscribed to by public health people. However, there are several important responsibilities which by theory and experience fall to public health. In all situations, even in countries with highly competent and well developed public works organizations, it is incumbent upon public health agencies to stimulate undertakings that have an important relationship to health. Public works departments should welcome the helpfulness of such suport, and usually the closest co-operation exists. It is also logical that public health agencies should establish and enforce standards for the service and performance of systems and plants that have a bearing upon the health of the people. Since improper installations of enormous cost cannot well be condemned or torn down, it follows that public health organizations should have the responsability for the approval of the designs of all works of a sanitary nature. For the same reason public health agencies must continue to exercise general oversight over the operation of all such systems and plants.

In a word, the relations between public health and public works agencies should be co-operative and complementary. If such exists and is competent, the public works department should carry out its normal functions of engineering investigation, design and construction — sometimes operation. The health department should establish the need for the improvement and set the standards for its performance; it should assist the PWD in promoting the works with officials and the public; it should approve the plans for the undertaking in those respects which relate to health; and it should have the duty of requiring that the works be operated in a safe and sanitary manner. Only in the absence of a qualified public works organization should the health department itself, with its own forces or by the use of consultants, assume the responsibilities of design and construction.

However, in every country it will be found that sanitary works of the smaller types, such as individual wells, privies, septic tanks, composting yards, public baths and laundries, and the installations serving schools and similar institutions will usually be too small and too dispersed to be of interest to public works departments or to commercial firms. Moreover, such projects usually require the educational preparation, and often the application of the police power of health departments. In most cases they involve the contribution of labour or materials by the people. The proper use and maintenance of the facilities must be rigidly enforced. For all of these reasons it becomes necessary in most places for health agencies to assume the entire responsibility for these simpler types of physical improvement.

DEVELOPMENT OF PERSONNEL

In sanitation, as in all other sectors of public health, the competence of personnel is of first importance. The absence of qualified staff is the most serious deficiency of today, and in sanitation there is perhaps a greater scarcity than in any other field. To make matters worse, the greatest dearth is in areas where the need for sanitary improvements is most severe. For years the Rockefeller Foundation, and in later times organizations such as WHO and ICA, through fellowship grants, have trained a few sanitary engineers in several countries. Those who found placement in the profession are now the backbone of sanitation units of health agencies, but they do not go very far in supplying the international and national needs. Organizations such as WHO require a very high level of competence in men able to serve as advisers to governments, as teachers in universities and as leaders of field projects. An attempt is made to recruit such staff in the highly competitive market of the western countries, which is the only present source of adequately trained personnel. This recruitment is very difficult and uncertain, and often posts remain vacant for long periods. At best the tenure of such persons is short, and often there are problems of adaptation, climate, language, politics, or the education of children which impair both happiness and efficiency.

The co-operating countries usually have an even more difficult time furnishing qualified counterpart personnel on projects or in staffing their newly organized sanitation units. Often acceptable persons are simply not available. Fellowships for foreign training do not always provide a solution. In addition to the normal difficulties and pitfalls of these fellows who go abroad, some governments are finding that the enticements of Cupid or the propositions of foreign commercial organizations are causing an attrition of more than 50 per cent. in those returning home to work.

All of these conditions, and more, argue for the same solution to this vexing problem: train the sanitation personnel close to home. Such a plan would avert many of the difficulties now encountered and would produce more people better suited to do the practical tasks at hand, who could work with greater efficiency and contentment. Education on a collegiate level would be conducted by either regional or national institutions, depending upon circumstances, while the lower level training would be given in numerous training centres, strategically located, for shorter periods of time. This plan would not mean a cessation of international fellowships for sanitary engineers and sanitary scientists. Selected persons who have won the right by demonstrated merit, and who promise to occupy positions of leadership in health agencies and universities, would go abroad in fewer numbers than heretofore for the best obtainable technical education, and then return to posts that would be waiting for them. Nor would this programme of localized education stop the necessity of international recruitment by WHO and similar agencies. Highly qualified persons would still be required to fill the key positions of advising governments, guiding programmes, teaching in universities and in developing the national personnel.

GENERAL DISCUSSION

Dr. Fernando M. de Bustamante — Sanitary Engineering substituted as the title of a program for environmental sanitation, implies an interest on the health of people on a wider and larger field, I am in agreement with Dr. Baity as to the problems of recruitment of personel and the availability and permanence of young trained people in key sanitary engineering positions. Infortunately this is something plaguing the training of public health doctors, nurses and other people when the financial rewards are less than in private practice or ocupation. Engineers and doctors need to work closely together and I would like to mention the environmental sanitation program in Mexico presented to the A.P.H.A. in Cleveland last November, where the environment is modified in the physical aspect in water supply, sewage disposal or latrine construction, house improvement opening windows, building furniture, etc. In the mental environment the attitude of people is improved as they have light in their houses; the food is cooked above ground, etc. In the social aspect, the comunity takes interest in all the improvements and even the streets are made different and pleasant.

Colonel Philip R. Beckford — Questions to Dr. Baity of WHO:

1) What is WHO position on the role of the sanitary engineer in radiological hygiene and industrial hygiene engineering?

2) Please review the progress of training of sanitary engineers since second world war in non western countries.

Eng. H. G. Baity — The responsabilities of WHO in the sanitary engineering aspects of radiological health have not yet been defined, but are being worked out in cooperation with the International Atomic Energy Agency.

In the field of industrial hygiene there is close cooperation between the WHO and the ILO.

Statistics are not immediately available on the number of sanitation posts which have been stimulated by WHO. In fact it would be impossible to differentiate between those resulting from the activities of WHO and other international agencies such as ICA. However, *in toto*, they now run into hundreds.

Much encouragement comes from the training and educational facilities which have been stimulated. About a dozen universities now offer graduate programes in sanitary engineering and a larger number of training schools and institutes are engaged in the training of other sanitation personnel.

ADENDA

ENSAYOS DE TERAPIA EN LA ENFERMEDAD DE CHAGAS AGUDA CON LA ASOCIACION DEL 8-(4-AMINO-1-METILBUTILAMINO)-6-METOXIQUINOLINA DIFOSFATO O DIFOSFATO DE PRIMAQUINA Y ACETATO DE CORTISONA [1]

(INFORME PRELIMINAR)

LUIS ESPINOZA [2]

La enfermedad de Chagas, Tripanosomiasis americana o Esquizotripanosis, sigue constituyendo hoy día un complejo problema terapéutico. Muchos compuestos químicos e incluso antibióticos han sido probados, sean en forma experimental que clínica (1, 2, 3, 4, 5, 6, 7, 8) con resultados siempre deficientes. Algunos éxitos iniciales obtenidos, fueron seguidos más tarde de ruidosos fracasos por las recidivas frecuentes que presentan los casos tratados.

El mejor conocimiento que se tiene hoy de su patologia y sobre todo de la biologia del Tripanosoma cruzi, han descartado en forma definitiva de su terapia diversas sustancias conocidas como esquizotripanocidas o tripanocidas, las que sin embargo, demuestran una acción eficaz en otras Tripanosomiasis, como por ejemplo en las africanas. En efecto se sabe perfectamente que el ciclo vital del parásito de Chagas difiere completamente de aquellos que siguen otras especies de Tripanosomas patógenos. Por este motivo, se piensa hoy con justa razón, que la acción terapéutica en la enfermedad de Chagas debe ser dirigida particularmente contra la forma tisular o leishma-

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[1] Trabajo realizado en el Departamento de Medicina Tropical del Instituto Nacional de Higiene, Guayaquil — Ecuador.

[2] Médico-Jefe encargado del Departamento de Medicina Tropical del mismo Instituto.

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nioide del parásito, por ser ella, en fin de fines, la causante de la patologia observada en esta enfermdad, más no contra la forma flagelar hemática de invasión, considerada por algunos autores sólo como un epifenómeno en el curso de la enfermedad.

Hoy se cree que el tropismo especial que posee el parásito de Chagas por ciertos tejidos mayormente evolucionados (haz de His, célula nerviosa, etc., etc.) sea determinada por razones de su carga eléctrica. En efecto y como lo pudo demostrar A. V. Szent-Giorgy (9) en un estudio de cataforesis practicado sobre diversos protozoarios, el Tripanosoma cruzi posee normalmente una carga eléctrica diversa a aquellas observadas en los Tripanosomas africanos, pués mientras el Tripanosoma cruzi es anódico, los Tripanosomas africanos son catódicos.

El hallazgo casual hecho por Séneca (10) del Tripanosoma cruzi en monos salvajes provenientes de la India en el curso de un estudio experimental sobre enfermedades reumáticas empleando altas dosis de Cortisona, vendria hasta cierto punto a confirmar esta teoria eléctrica, dado que es perfectamente conocido, que la Cortisona es en grado de invertir las cargas eléctricas normales moleculares de los organismos vivos. Ello tambien podria explicar en parte la parasitemia intensa que se provoca en un animal con infección chagástica crónica inaparente después de administradas ciertas dosis de Cortisona, y como diversos autores y nosotros mismos hemos tenido la oportunidad de comprobarlo en el laboratorio.

Muniz y Azevedo (11) en un importante estudio anátomo-patológico, pudieron comprobar como el Tripanosoma cruzi al penetrar en el organismo humano es capaz de ocasionar fenómenos de sensibilización a distancia sobre diversos órganos nobles como el corazón, hígado, pulmones, sistema nervioso, intestino, etc., provocando subsiguientemente al periodo de invasión, verdaderos choques anafilácticos en dichos órganos con formación de lesiones inflamatorias características que podrian favorecer en un segundo tiempo la anidación del parásito y su consecuente reproducción, ocasionando finalmente la degeneración y necrosis del tejido tocado.

La gravedad que asume en nuestro medio las complicaciones cardiacas y hepáticas (12, 13, 14, 15, 16) ocasionadas por este peligroso parásito, nos indujo a iniciar en un crecido número de enfermos un ensayo de tratamiento, el que decidimos orientarlo basándonos en las concepciones y hechos patogénicos que hemos aludido al comienzo.

Una particular acción esquizotripanocida del 8-(4-amino-1-metilbutilamino)-6-metoxiquinolina difosfato o difosfato de Primaquina contra el Tripanosoma cruzi fué demostrado en forma experimental por Goble (17) quien reportó efectos favorables de la droga en los ratones infectados.

En nuestra experiencia clínica decidimos asociar al difosfato de Primaquina el Acetato de Cortisona, tomando en consideración la acción beneficiosa de esta última sobre los estados inflamatorios agudos favoreciendo su resolución y la restitución ad-integrum de los tejidos afectados.

Un exaustivo estudio experimental en ratones blancos empleando esta misma asociación fué conducida por nosotros como acto previo a las experiencias clínicas. Estos resultados, que podemos catalogarlos excelentes, serán motivo de una próxima y documentada publicación, sobre todo anátomo-patológica, que en nuestro concepto, es la prueba de mayor valor en cualquier estudio que se efectue sobre la patologia de la enfermedad de Chagas, sea experimental o clínica.

Todos los pacientes en quienes fué seguido este tratamiento se trataron siempre de casos agudos, con tiempo de iniciación de la enfermedad variable desde los 5 días hasta los 32 días. Un particular cuidado fué observado para precisar la fecha exacta de cuando habia ocurrido el accidente infectante, que no siempre fué ocular, sino en partes diversas del cuerpo. Esta prolija medida seguida por nosotros, nos permitió a la vez observar que la parasitemia en nuestros pacientes siempre se presentaba escasa y poco persistente, sobre todo en aquellos pacientes en que fué iniciado con mayor precocidad el tratamiento y en muchos de los cuales aún ni siquiera presentaban parasitemia evidente ni síntoma alguno general de la enfermedad a excepción de una reacción local en el punto de penetración del parásito. Sin embargo, en todos fué siempre confirmado el diagnóstico por el hallazgo del Tripanosoma cruzi, en forma directa en aquellos casos que presentaban más de 15 días de iniciada la enfermedad, en los casos precoces fué siempre mediante el xenodiagnóstico practicado desde el primer día de consulta todos los días y en ocasiones 2 veces al día hasta el hallazgo del parásito. El control de los pacientes después del tratamiento siempre fué seguido con xenodiagnóstico al inicio cada mes, después cada 2 y 3 meses hasta un año o más de un año según los casos y las posibilidades con que contábamos.

Todos los enfermos desde su inicio fueron asilados en un Servicio Especial del Hospital de Enfermedades Infecto-contagiosas de la Dirección General de Sanidad en Guayaquil y sometidos a un estricto control de laboratorio, radiológico y electrocardiográfico cumplidos casi siempre a repetición durante todo el tiempo de su hospitalización que siempre fué prolongado a 6 meses como mínimo, seguidos posteriormente a domicilio por un tiempo aún mayor.

Dado lo voluminoso de la documentación que poseemos de cada caso y el deseo nuestro de presentar un estudio terapéutico en esta enfermedad después de una observación de por lo menos tres años después de efectuado el tratamiento, nos ha inducido a seleccionar 10 casos, que por ser los mas antiguos y completos que tenemos, para hacer una primera comunicación aprovechando las próximas reuniones del VI Congreso Internacional de Medicina Tropical y Paludismo a reunirse en Lisboa, proponiéndonos con esto llamar la atención de los especialistas hacia nuestro ensayo terapéutico en esta terrible enfermedad, la cuya gravedad ninguno puede negar y cuyo tratamiento siempre era nulo. Sólo queremos hacer presente que nuestro tratamiento se ha mostrado siempre bien tolerado por adultos y niños, sin ninguna manifestación de intolerancia o efectos secundarios de importancia, obteniéndose en cambio manifestaciones clínicas y de exámenes de laboratorio que han demostrado siempre una aparente curación definitiva de la enfermedad, como se pueden evidenciar en los electrocardiogramas que presentamos de cada enfermo, radiografias del corazón, xenodiagnósticos y reacciones de fijación del complemento. Es indudable que en una enfermedad de la tendencia crónica como es la Tripanosomiasis americana y el polimorfismo de sus formas clínicas siempre dejan una incógnita a cualquier estudio sobre terapia de la enfermedad, por esto son nuestros deseos esperar a hacer nuestro reporte definitivo después de por lo menos 3 a 5 años que podamos observar nuestros pacientes.

Los detalles de la edad, posologia y resultados de exámenes de control de estos 10 primeros casos que presentamos los hemos resumido en un cuadro esquemático que acompaña esta información preliminar.

				Tratamiento				Resultados obtenidos		
Enfermo	Edad	Sexo	Peso en lbas.	Dia de iniciación	Duración en días	Cantidad total		Cuadro clin. y tiempo de control	R. F. C. de control	Xenodiagnósticos de control
						Dif. de Primaq. (mg.)	Acet. de cortisona (mg.)			
L. S.	31 A.	F	130	14	15 cont.	450	900	Excelentes 18 meses	XII–12–56 : Pos III–25–57 : Neg. V– 5–57 : Neg.	IV– 6–57 : Neg. XII–16–57 : Neg. V– 5–58 : Neg.
N. P.	13 A.	F	96	16	16 cont.	400	800	Excelentes 16 meses	XII–28–56 : Ant. VI–26–57 : Neg. IV–14–58 : Neg.	VI–26–57 : Neg. XI– 5–57 : Neg. IV–14–58 : Neg.
Y. V.	17 A.	F	105	10	29 repartidos en 3 etapas	875	1350	Deficientes 9 meses	VIII–10–57 : Neg. XII–28–57 : Pos. VII–12–58 : Neg.	I–15–57 : Neg. IV–16–57 : Neg. VII– 7–58 : Neg.
R. A.	25 A,	M	120	12	15 cont	550	1100	Excelentes 15 meses	IV–15–57 : Pos. IX–12–57 : Neg. V– 8–58 : Neg.	IX–12–57 : Neg. XII– 5–57 : Neg. VII– 8–58 : Neg.
N. D.	36 A.	F	134	15	16 cont.	550	1100	Excelentes 15 meses	IV–12–57 : Pos. VIII– 8–57 : Neg. V–20–58 : Neg.	VIII–10–57 : Neg. XII–12–57 : Neg. V–20–58 : Neg.
A. V.	27 A.	F	116	17	16 cont.	500	1000	Excelentes 17 meses	II–19–57 : Pos. XI–26–57 : Neg. VI–14–58 : Neg.	VIII–26–57 : Neg. XI–25–57 : Neg. V–15–58 : Neg.
L. Z.	15 A.	F	205	10	27 en 2 etapas	675	925	Dudosos 10 meses	VII–30–57 : Pos. XII–16–57 : Neg. VII–20–58 : Neg	IX–16–57 : Neg. I–15–58 : Neg. VII–19–58 : Neg.
V. H.	20 A.	M	126	5	16 cont.	475	1050	Excelentes 16 meses	III–22–57 : Pos. VIII–27–57 : Neg. V–15–58 : Neg.	IV– 6–57 : Neg. VIII–16–57 : Neg. V–15–58 : Neg.
J. M.	12 A.	F	86	32	16 cont.	400	800	Excelentes 17 meses	II–18–57 : Pos. X–22–57 : Neg. V–21–58 : Neg.	VIII– 4–57 : Neg. XII– 6–57 : Neg. V–16–58 : Neg.
N. G.	5 m.	F	20	12	12 cont.	120	240	Excelentes 12 meses	IV– 3–57 : Pos. X– 8–57 : Neg. V–12–58 : Neg.	VI– 6–57 : Neg. X– 7–57 : Neg. V–12–58 : Neg.

RESUMEN

De un estudio mayor sobre tratamiento de enfermedad de Chagas aguda utilizando el difosfato de Primaquina y acetato de Cortisona conjuntamente, fueron seleccionados diez casos que reunian una documentación clinica bastante completa y cuyo tiempo de observación se prolongó a más de un año.

La iniciación del tratamiento fué precoz en uno de ellos (5 días) y tardia en los 9 restantes (10 a 32 dias).

Cuando se inició el tratamiento, dos de los enfermos presentaban alteraciones cardiacas y hepáticas graves. Cinco de ellos tenian trastornos cardiacos moderados solamente, y tres restantes no presentaban signos de trastorno de ningun órgano aparentemente.

Ocho de los pacientes recibieron su tratamiento en una sola serie continua (12 a 15 dias) con un total que osciló entre los 450 a 550 mg de difosfato de Primaquina y 900 a 1 100 mg de acetato de Cortisona en los adultos, y entre 120 a 400 mg de difosfato de Primaquina con 240 a 800 mg de cortisona en niños de primera y tercera infancia.

Dos de los enfermos recibieron su dosis total en tres series (27 a 29 dias) con una cantidad que fluctuó entre los 675 y 875 mg de difosfato de Primaquina y 925 a 1 350 mg de Cortisona.

Las drogas fueron administradas en todos los enfermos cada 24 horas y siempre por via parenteral.

Controles hematológicos practicados rutinariamente indicaron una baja discreta de los niveles normales en todos los casos que fueron corregidos exitosamente con sustancias antianémicas, especialmente sulfato ferroso.

No hubieron manifestaciones tóxicas en ninguno de los enfermos ni tampoco se presentaron reacciones secundarias. El contaje de los leucocitos no se modificó mayormente ni hubo manifestación de agranulocitosis. La investigación de glucosa en la orina fué siempre negativa. El control del peso corporal en todos ellos no indicó alteración alguna, asi como tampoco de la presión arterial. A todos ellos se les prescribió dieta hiposódica durante el tratamiento y se les administró cloruro de potasio diariamente en poción.

Controles electrocardiográficos, radiológicos, practicados a todos los enfermos con posterioridad a su tratamiento ha demostrado una completa restitución a la normalidad de sus órganos afectados. De la misma manera pruebas de R.F.C. y xenodiagnóstico posteriores al tratamiento fueron siempre negativos.

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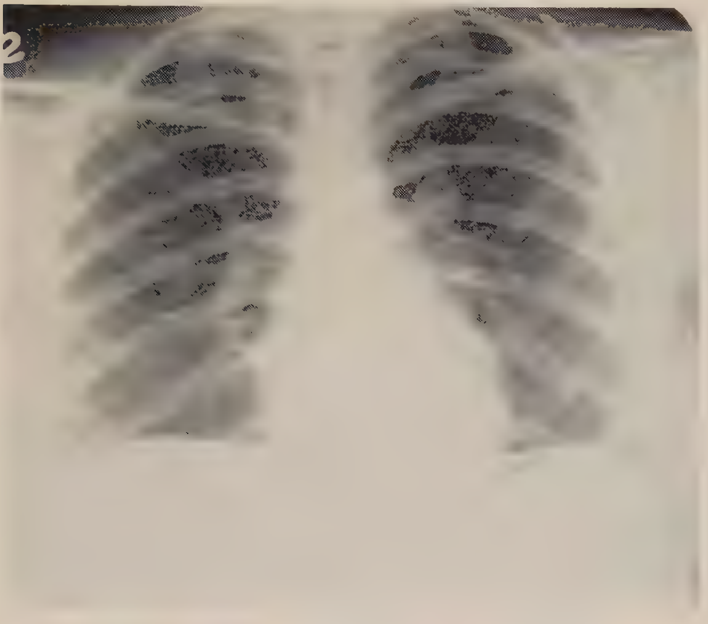
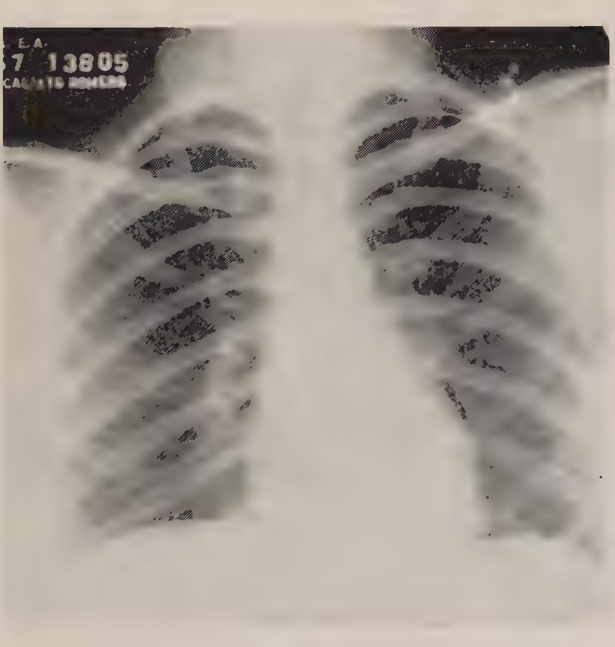
CLAVE

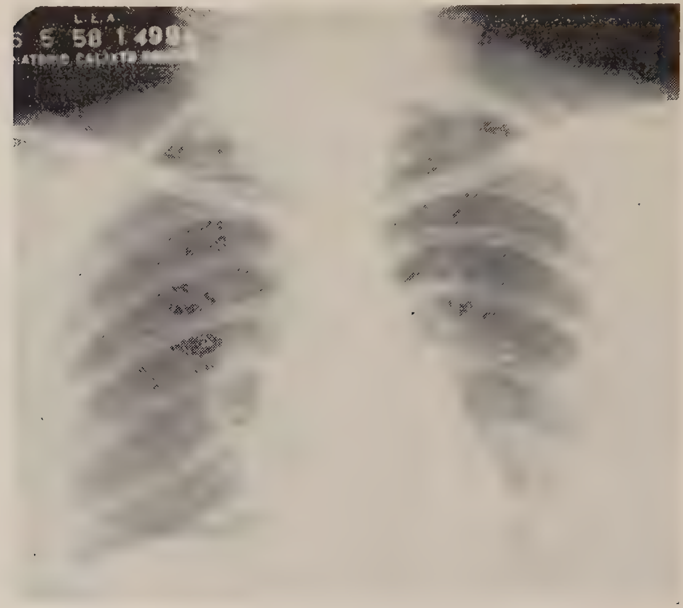
R.F.C. — Reacción de fijación del complemento
Xeno. — Xenodiagnóstico
cont. — continuos
Antic. — Anticomplementario
Neg. — Negativo
Pos. — Positivo
A. — Años
m. — meses
F. — Femenino
M. — Masculino



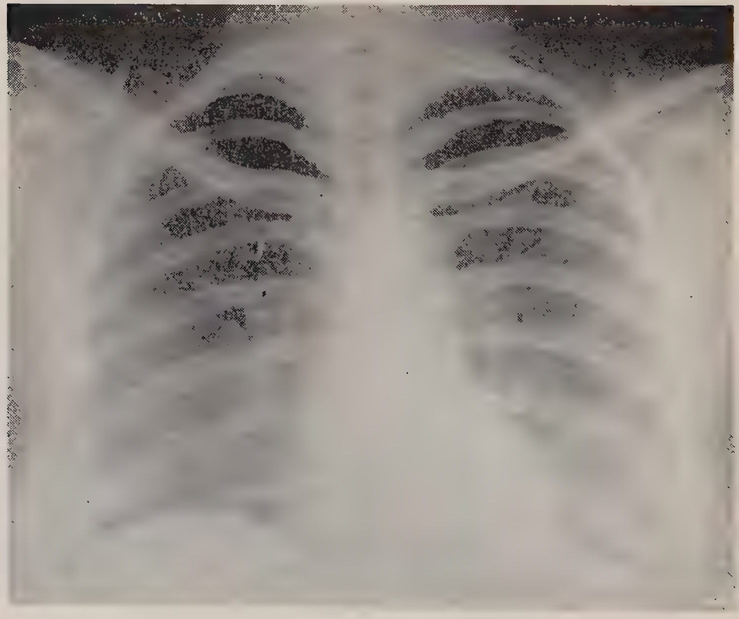
Paciente L. S. — Al inicio del tratamiento



Paciente L. S. — 3 meses después del tratamiento



Paciente L. S. — 9 meses después del tratamiento



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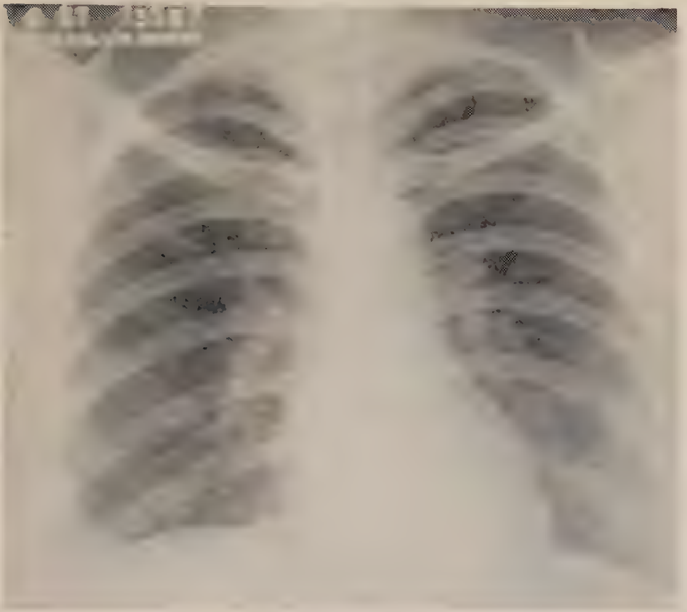
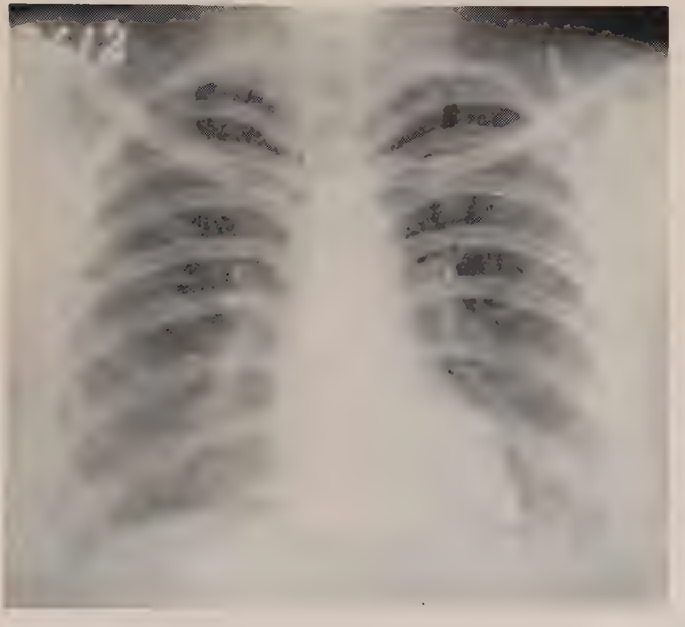
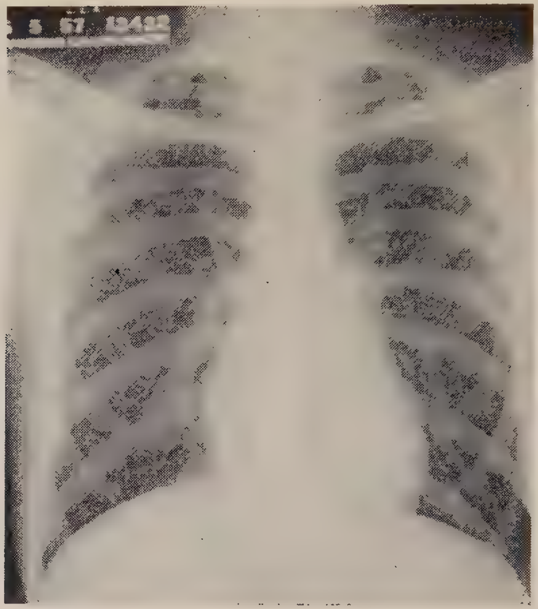


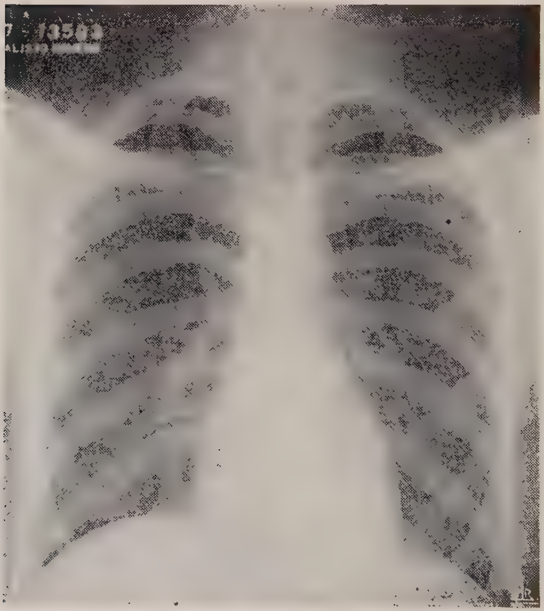
Paciente N. P. — 2 meses después del tratamiento



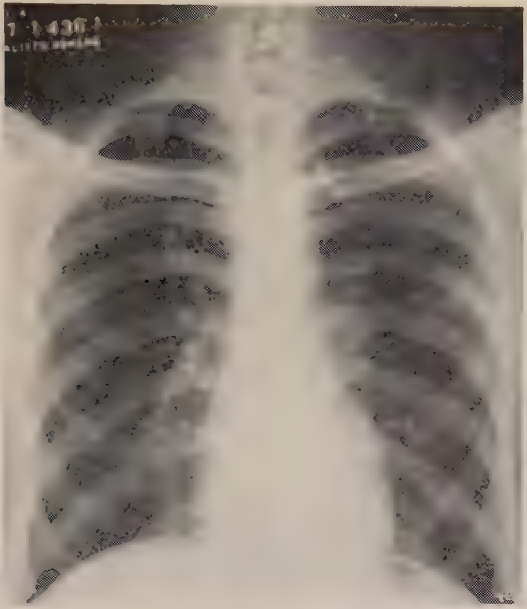
Paciente N. P. — 13 meses después del tratamiento



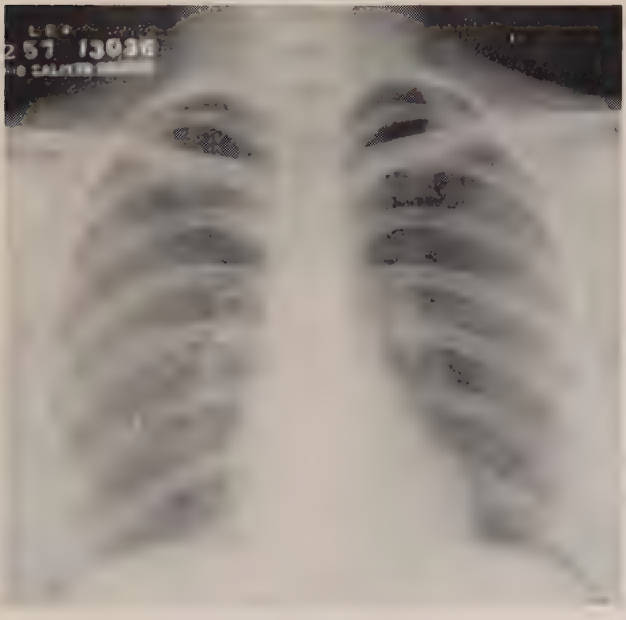
Paciente Y. V. — Al inicio del tratamiento



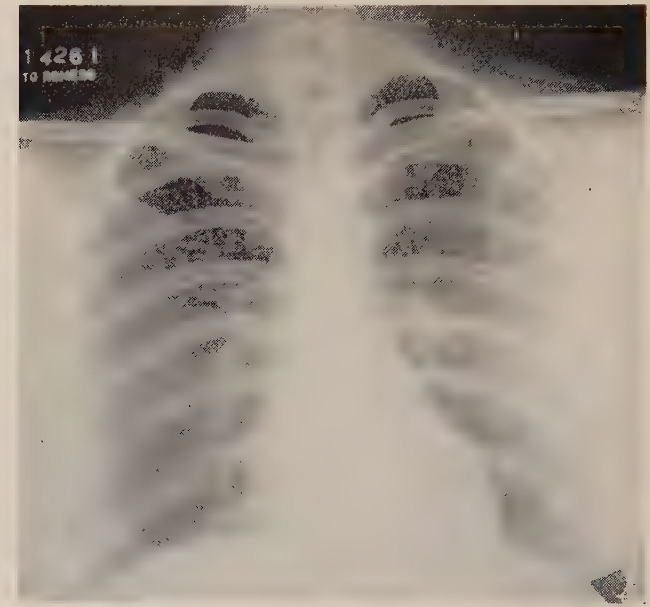
Paciente Y. V. — 3 meses después del tratamiento



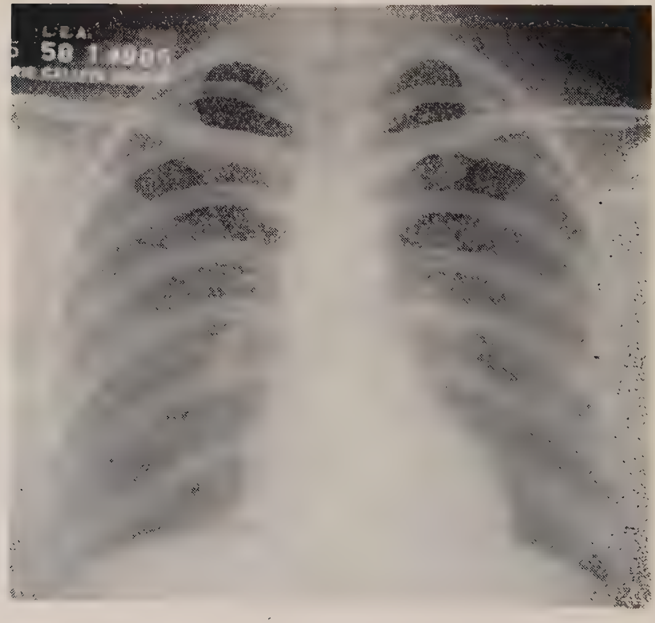
Paciente Y. V. — 8 meses después del tratamiento



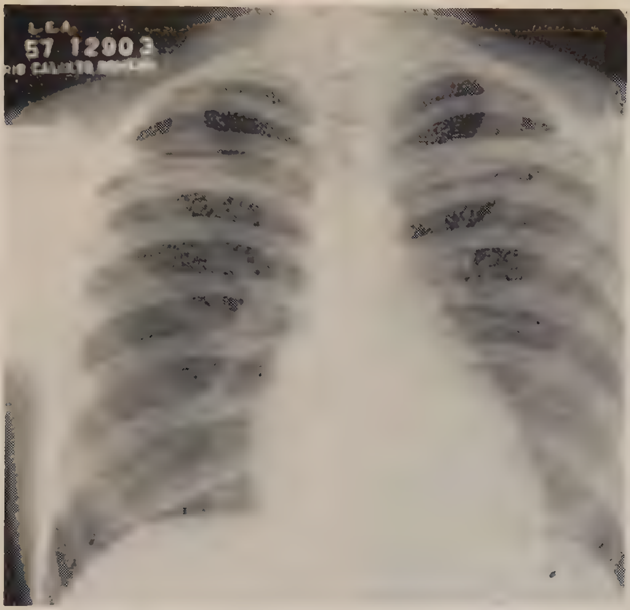
Paciente R. A. — Al inicio del tratamiento



Paciente R. A. — 3 meses después del tratamiento



Paciente R. A. — 15 meses después del tratamiento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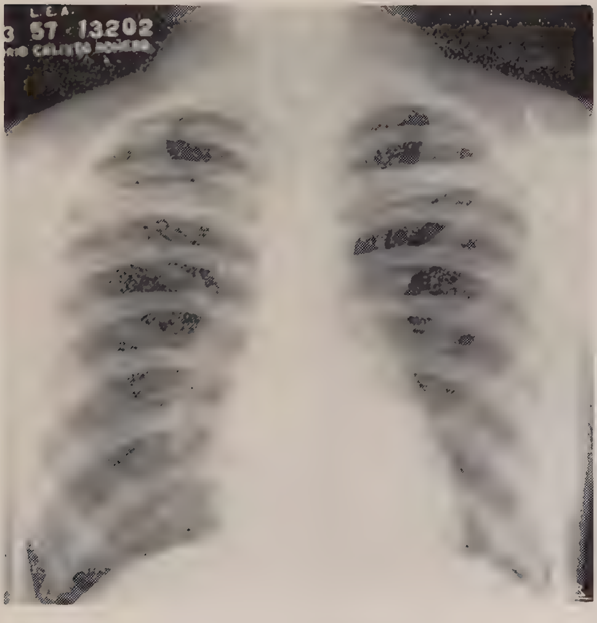
Paciente N. D. — Al inicio del tratamiento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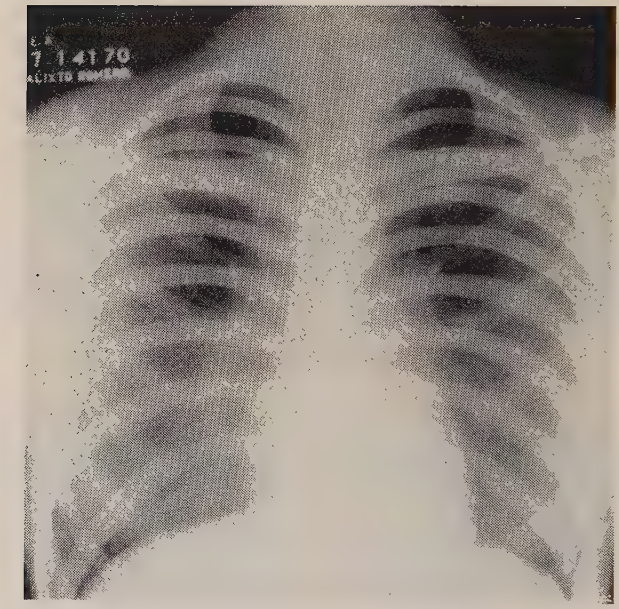

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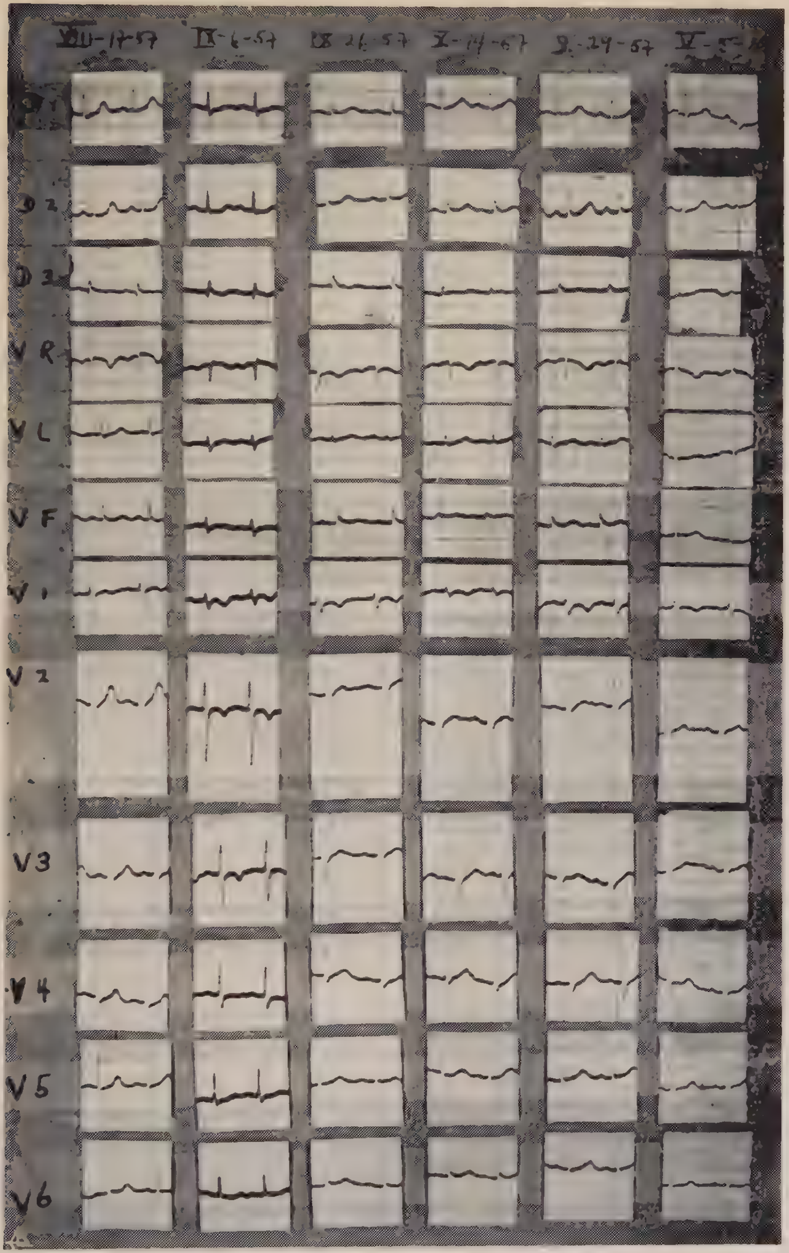
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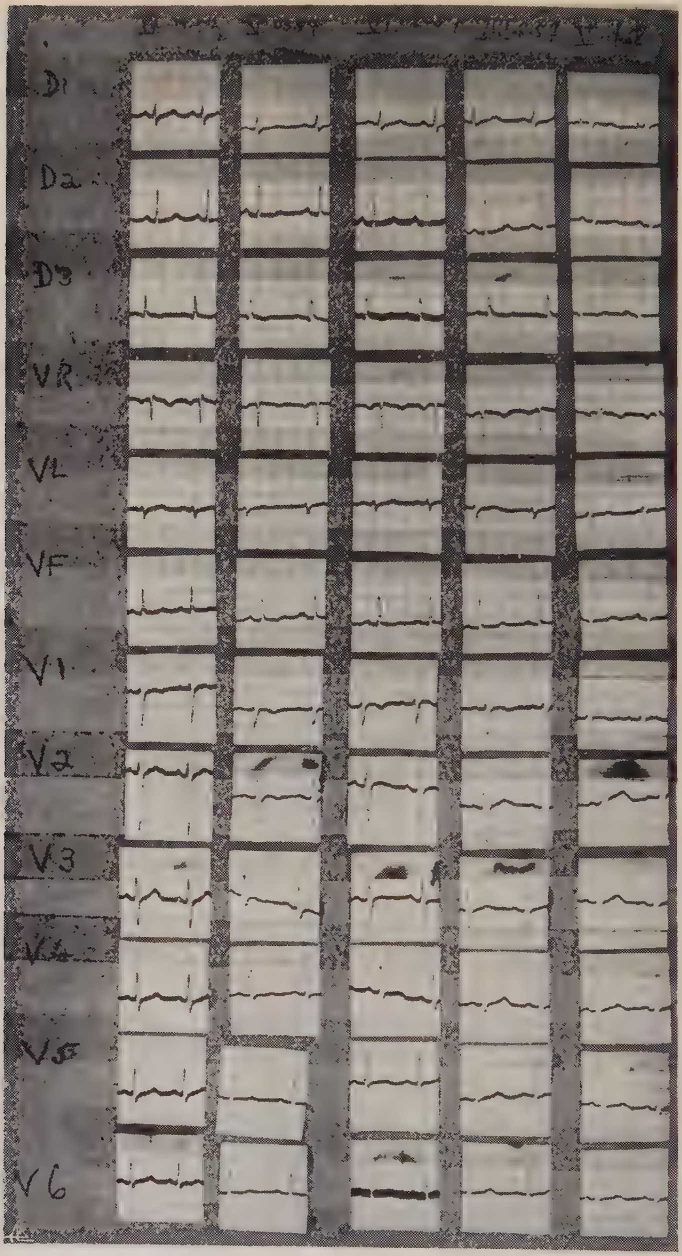
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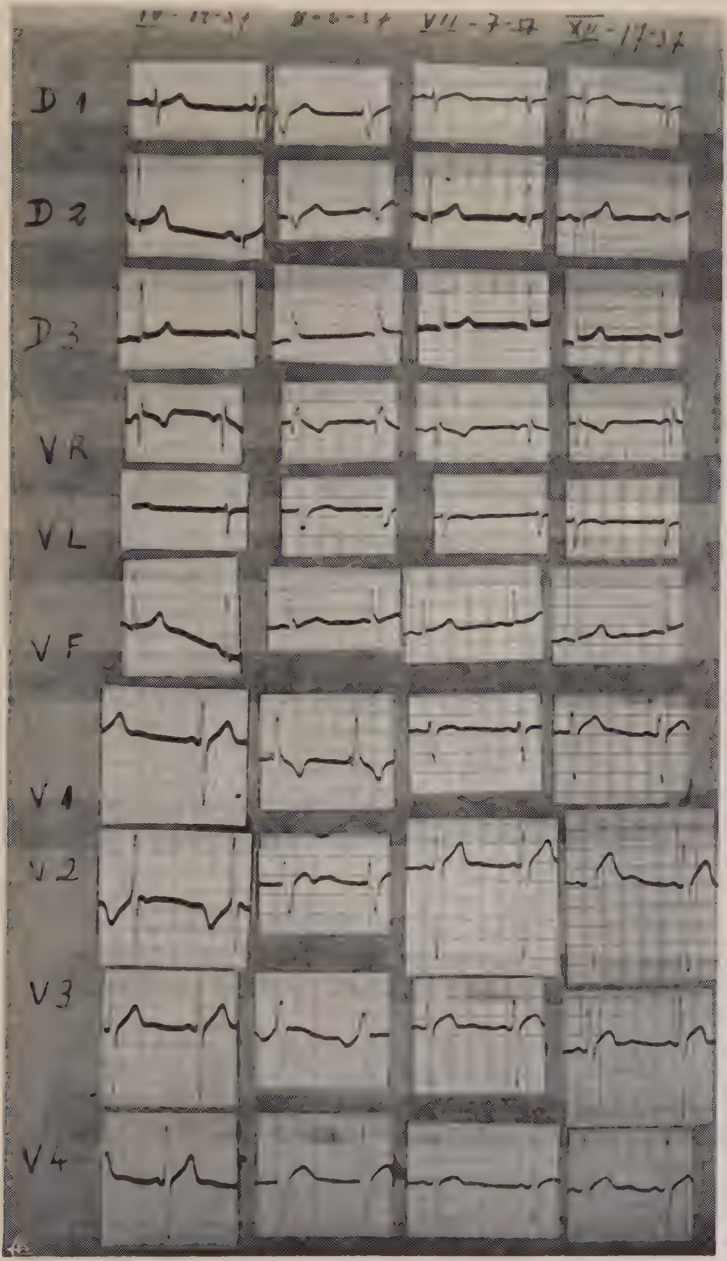
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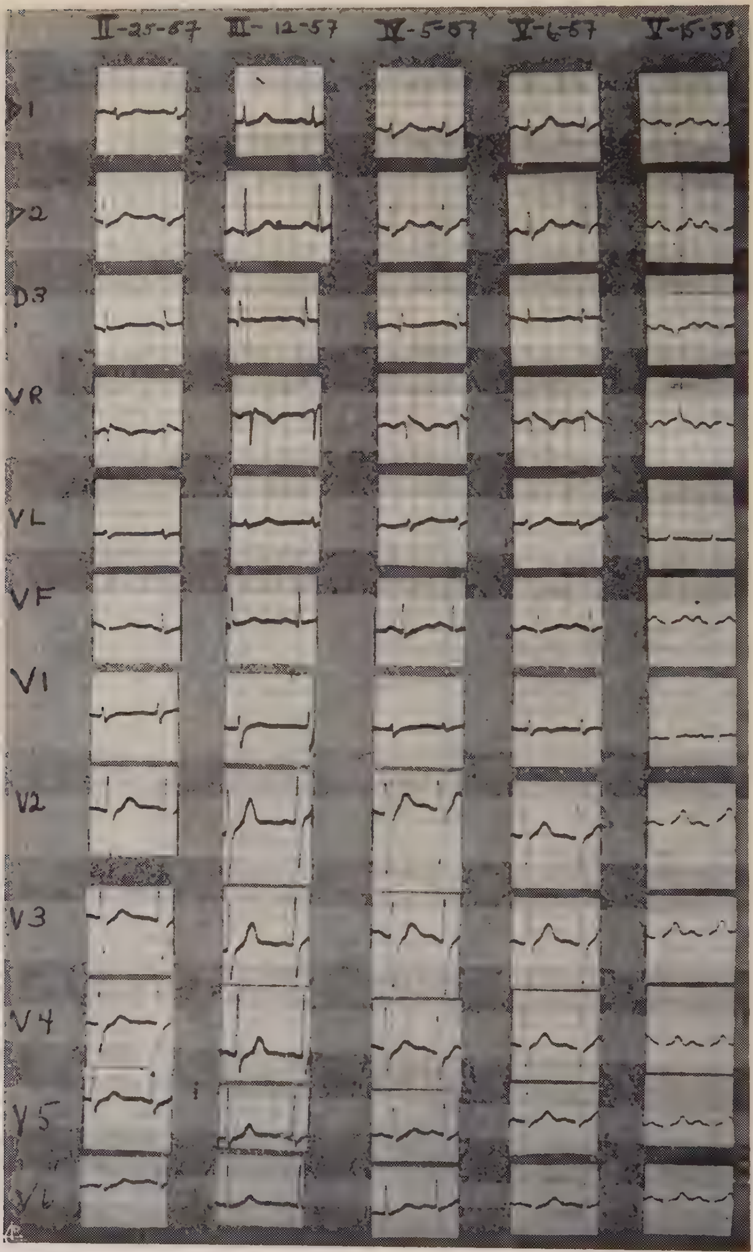
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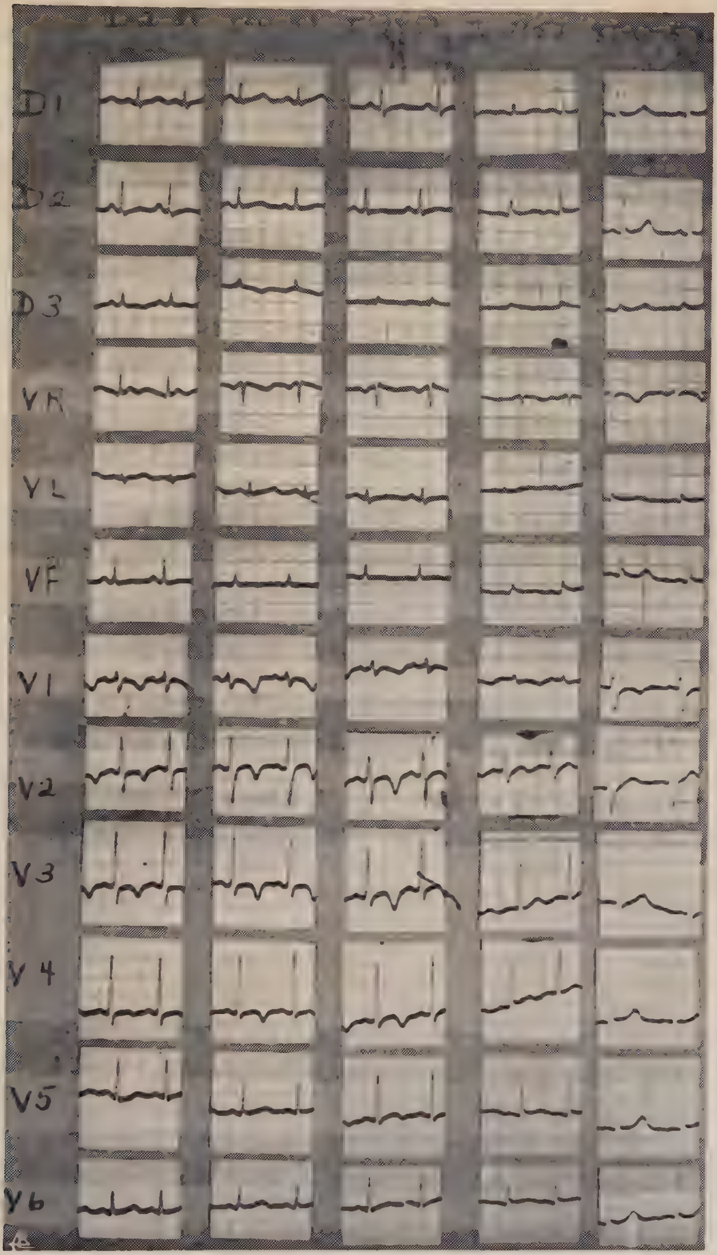
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